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THE Journal OF THE Kansas Medical Society

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1973

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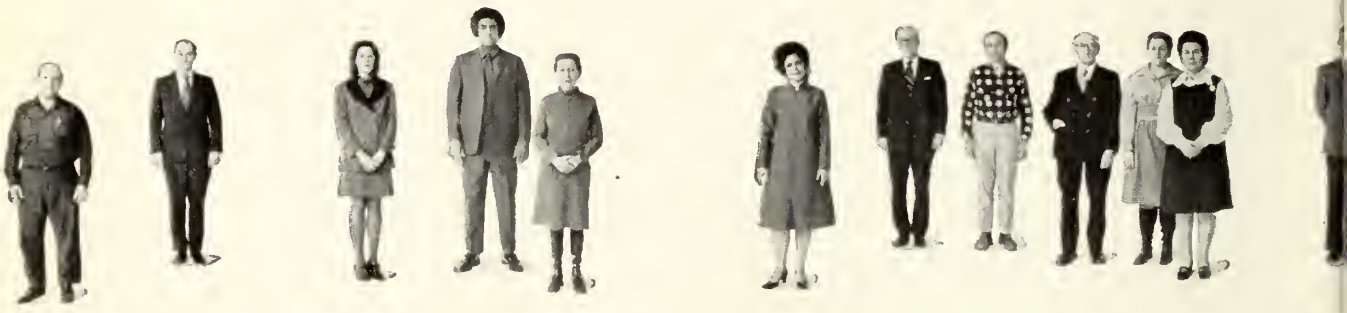

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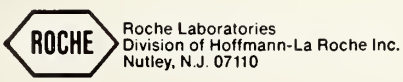

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Volume LXXIV, Number 7—July, 1973

The JOURNAL of the KANSAS MEDICAL SOCIETY

Contents for July

Scientific Articles

Pulmonary Nocardia Asteroides: Successful Treatment with Sulfadiazine—Robert F. Byrne, M.D., F.C.C.P. and David Givner, M.D., F.A.C.P., Wichita . . 265
Gastroduodenal Visualization: Fiberoptic Esophagogastroduodenoscopy—Nirmal S. Mann, M.D., F.R.C.P., Topeka 269
Aortic Valve Replacement: Prosthesis Related Complications—Duncan A. Killen, M.D., John P. Sutton, M.D. and Harold A. Collins, M.D., Kansas City, Missouri 273
Source and Significance: Midsystolic Clicks and Late Systolic Murmurs—Scott Allen Lerner, M.D., Kansas City, Kansas 276

Student Thesis

Pseudomonas Pneumonia—Martin S. Hirsch, M.D., Kansas City, Missouri . . 280

Miscellaneous

Advertisers 19
AMA Committee on Health Care of the Poor 290
Amphetamines & Growth 14
Announcements 296
Classified Advertisements 19
Convention Glimpses 293
D. C. Line 294
Editorial Comment 286
Hyperlipoproteinemia Course 288
Information for Authors 268
Kansas Morbidity Incidence Report 16
Kansas Press Looks at Medicine 17
Month in Washington 12
New Members 19
Obituaries 289
Parents in Day Care 10
Personalities in Medicine 292
Physicians in Politics 294
President's Message 285
Speakers Available 8, 16
Vox Dox 287
Woman's Auxiliary 295

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Scientific ARTICLES

Pulmonary Nocardia Asteroides

Successful Treatment With Sulfadiazine

ROBERT F. BYRNE, M.D., F.C.C.P.* *and*
DAVID GIVNER, M.D., F.A.C.P.,* *Wichita*

The purpose of this article is to review the pertinent laboratory, clinical, and therapeutic aspects of pulmonary nocardiosis, and to report a case successfully treated with sulfadiazine. Comparable blood levels of sulfadiazine were prepared in media, and the growth of Nocardia from the patient was completely inhibited. Three months after the patient was cured clinically from his nocardiosis, he had a stroke and died. Autopsy showed residual pulmonary fibrosis with no evidence of any active pulmonary disease.

Bacteriology

NOCARDIOSIS is a chronic disease characterized by visceral suppurative and granulomatous lesions. Nocardia asteroides, which has been classified as a fungus in the past, is now considered a member of a separate group called Actinomycetales, which also includes the Actinomycetaceae, Mycobacteriaceae, and Streptomycetaceae. It is a solid organism that grows as a mold in nature and in humans, and appears in tissues as thin, occasionally branching hyphae that easily fragment and take the acid-fast stain. Since Nocardia asteroid is bacillary and partially acid-fast, it may be confused with Mycobacterium tuberculosis when seen on smears. *Table 1* shows a classification of the Actinomycetales.[1]

Nocardia can be grown from sputum following concentration with 4 per cent sodium hydroxide and neutralization with 4 per cent hydrochloric acid. Nocardia grows aerobically on Sabouraud's agar, blood agar, or 7-H10 Middlebrook media, and colonies will appear within four to six days. It is frequently granular and powdery, and the color varies from white to deep orange. Microscopically, there are delicate branching, intertwining filaments which are gram-positive, and may or may not be acid-fast.

The various pathogenic species of Nocardia are Nocardia Africana, the pink to red colony on culture; Nocardia asteroides, a yellow to orange colony; Nocardia Brasiliensis, which has a tan to yellow to pink growth on culture; Nocardia gibsanii, cream-colored; Nocardia leishmanii, white to pink; Neisseria madurae, a buff to pink growth on culture; Neisseria preteriama, buff to pink in color; and Nocardia ragoonensis, which has a cream to yellow-brown color. Gordon[2] described Nocardia dassonvillei, which is a macroscopic replica of Streptomyces griseus.

Diagnosis is established by culturing the organisms and the various cultural characteristics which can be described as acid-fast, aerobic, growing a yellow to orange type colony, and sometimes a white

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TABLE 1
CLASSIFICATION OF ACTINOMYCETACEAE
Relationship of Actinomyces and Nocardia sp. to true bacteria and Mycobacterium tuberculosis.

SCHIZOMYCETES (bacteria)
Eubacteriales (true bacteria): Produce no mycelium and no branching.
ACTINOMYCETALES
Mycobacteriaceae: Produce branching but no mycelium. Acid-fast. Aerobic.
Actinomycetaceae
Actinomycetes: Produce branching mycelium. No spirals. Segments to bacillary and coccoid forms. Nonacid-fast. Anaerobic.
Nocardia: Produces branching mycelium. No spirals. Segments to bacillary and coccoid forms. Somewhat acid-fast. Aerobic.
Streptomycetaceae: Produce branching mycelium without segmentation. Spirals are formed. Nonacid-fast. Aerobic.

fluffy type colony. Ordinary bacilli will not grow on media containing the broad spectrum antibiotics, such as chloromycetin. Nor will they grow following concentration with a 4 per cent sodium hydroxide and neutralization with 4 per cent hydrochloric acid media. No serological or skin tests are available for nocardiosis.[5]

Incidence

Mycoses are reported on an optional basis to the Center for Disease Control, but reporting procedures vary considerably. Therefore, it is not possible to piece together a national picture of the mycoses. In 1970, the US Public Health Service listed nocardiosis as the rarest mycotic infection in the United States, with three cases from Minnesota and one case from Missouri.[3]

Biochemical Reactions

A few of the biologic properties of the aerobic Actinomycetes remain constant when several strains of the same species are tested. It has been found that the genera Streptomyces and Nocardia could be separated by the following reactions: hydrolysis of casein, decomposition of tyrosine, and acid production from lactose, maltose, xylose, and mannose. These reactions were positive for Streptomyces, but negative for Nocardia. Pathogenic species within these genera can be separated by additional tests (McKinnon and Artavageytia-Allende, 1956; Bojalil *et al.*, 1959; Mariat, 1962).[4]

Clinical Course

Nocardiosis is world-wide in distribution and equally distributed between the sexes. The organism enters the human host by way of the respiratory tract or the skin. The clinical picture of pulmonary infection varies from mild to severe, and when severe it may include high fever, chills, and a course generally indistinguishable from that of acute bacterial pneumonia.

Nocardiosis involving the lungs may follow or be associated with prolonged treatment with cortisone, ACTH, anti-leukemic drugs, the terminal stages of malignant disease, particularly the malignant lymphomas, alveolar proteinosis, and it may be present as a part of an unusual course of illness in a patient with diabetes. In the case which we are reporting, the patient was an alcoholic.

The patient may present with an acute pneumonia, with the white blood count (WBC) as high as 50,000, and all of the other acute symptoms of pneumonia; he may present with a chronic illness with low fever, flu-like symptoms, cough, and WBC of 11,000-16,000. X-ray may show an acute pneumonia, a granulomatous type of lesion, or a diffuse infiltration. In the more chronic form, there may be single or multiple cavities prevalent to simulate tuberculosis or other cavitating lesions, such as the systemic fungi.

Treatment

In pulmonary nocardiosis, the treatment of choice at the present time is sulfadiazine. In the past, some of the other sulfas, such as gantrisin, triple sulfas, and sulfamerazine have been used successfully. A sulfonamide with streptomycin, or a tetracycline with cycloserine, may be used in more serious cases.[6] In treatment with sulfadiazine, high blood levels (15-20 mg/%) should be maintained for 4-6 weeks during hospitalization of the patient; lower blood levels (9-12 mg/%) should be maintained for at least six months after the patient's discharge.[4]

Case Report

A 63-year-old white male was admitted September 10, 1970, with the chief complaint of chills, fever, and cough of one day's duration. He had known emphysema since 1963, and had been unable to work for three years due to shortness of breath. He had not felt well one month prior to admission and had lost 14 pounds of weight. Temperature was 100F (37.8C) on admission; pulse rate was 100 beats per minute. The patient was a pale white male who appeared chronically ill. His chest showed evidence of emphysema with increased anteroposterior diameter with some kyphosis, poor expansion, hyperresonance to percussion, and distant breath sounds. There were no rales noted on admission. Other physical signs were normal.

X-ray of the chest on admission *(Figure 1)* showed heavily mottled inflammatory infiltrates, dif-

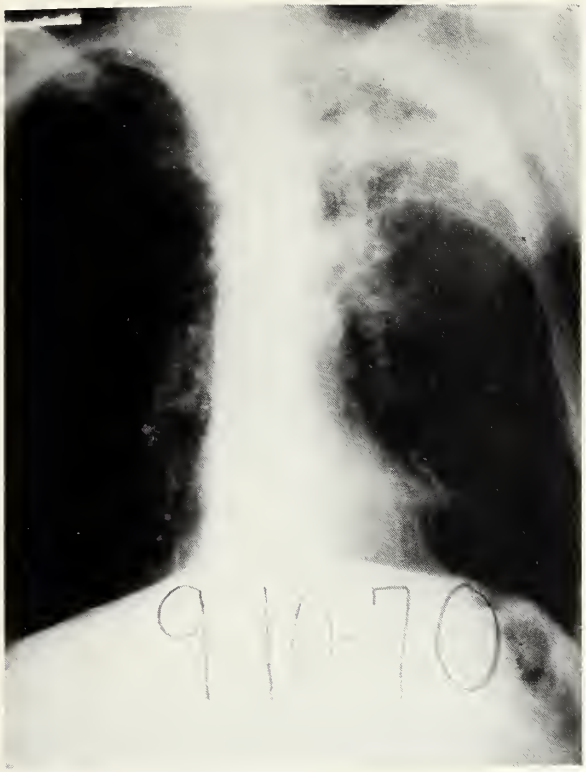

Figure 1. Chest x-ray at onset of present illness.

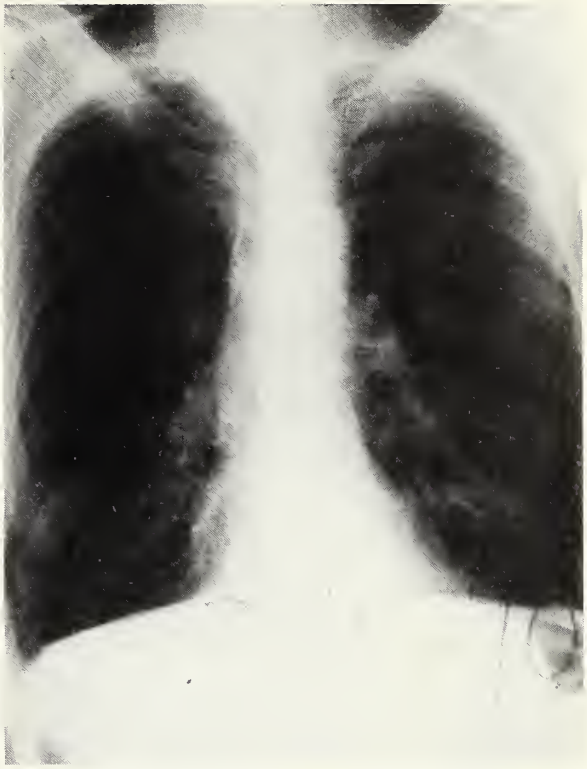

Figure 2. Chest x-ray 6 months prior to present illness.

fusely scattered throughout the upper lobe of the left lung, with multiple areas of numerous radiolucency indicating multiple cavities. Chest x-ray taken on March 18, 1970 *(Figure 2)*, had not shown any of this infiltrate or cavitary lesion, but had shown pulmonary emphysema, which again was seen on this admission. Hemoglobin was 10.5 gm/100 ml; WBC 16,400 cu mm, with 70% neutrophils, 2% juveniles, 7% bands, 15% lymphocytes, and 10% eosinophils. Results of blood glucose, BUN, urinalysis, and blood serology were normal. The intermediate purified protein derivative (PPD) skin test was 20 mm; histoplasma skin test 10 mm; coccidiomycosis skin test was negative. Culture of the sputum grew out of heavy white fluffy colony on Sabouraud's media and on 7-H10 Middlebrook media.

After the diagnosis was established by repeated positive cultures on October 6, 1970, a therapy of sulfadiazine, 3 gm every 6 hours, was started. This regimen was continued until January 29, 1971, and then decreased to 1½ gm every 6 hours through September 27, 1971.

Blood levels on sulfadiazine varied from 31 to 37 mg/per cent on the 12-gm daily dose, and 22 mg/per cent on the 6-gm daily dose. Culture media was prepared containing 25 mg sulfadiazine/100 cc to compare with the same percent basis as the blood. This completely inhibited the growth of Nocardia, although it would grow on media which did not contain the sulfadiazine.

The patient's body temperature varied from 99F (37.2C) to 100F (37.8C) daily, until after six weeks of sulfadiazine, when it stabilized and remained normal.

Three months after the treatment was begun, the patient had regained his lost weight, hemoglobin rose to 15.6 gm, WBC became normal, and the eosinophil count, which had varied from 9 to 15 per cent, became normal. The sputum cultures remained positive until six months after treatment was started, when they became negative and remained negative. Clinically, he was much better within one month.

A series of chest x-rays showed no change for the first six weeks. Ten weeks after treatment was started, a chest x-ray showed that the cavitation had disappeared and the infiltration had lessened considerably. After three months *(Figure 3)* the area had shrunk to a small area of fibrosis and remained stable *(Figure 4)*.

This patient suffered a cerebrovascular thrombosis three months after completion of his sulfadiazine therapy for pulmonary nocardiosis. Spinal tap showed clear fluid cell count of 2, and negative culture for nocardiosis. Autopsy showed some fibrosis in the previously involved area of the left lung, but no microscopic evidence of nocardiosis was present.

Figure 3. Chest x-ray 3 months after sulfadiazine therapy started.

Figure 4. Chest x-ray at completion of sulfadiazine therapy (11 mos. of treatment).

Acknowledgement

Miss Sylvia Bays for the bacteriology work.

(Continued on page 288)

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Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

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Gastroduodenal Visualization

Fiberoptic Esophagogastroduodenoscopy

NIRMAL S. MANN, M.D., F.R.C.P.,* ***Topeka***

GASTROSCOPY and the use of gastrocamera were quite common in Japan in the mid-sixties. Only very few gastrocameras were in use in the United States at that time. Since then, a tremendous increase in the interest and use of fiberoptic instruments for examination of the upper gastrointestinal tract has been noted in this country. Most endoscopists in Japan make routine use of the gastrocamera.[1] Today, many types of fiberscopes and gastrocameras are available; their safety and usefulness in the diagnosis and followup of lesions of the upper gastrointestinal tract have made them popular, even in small hospitals. While in the past there have been different instruments for the examination of esophagus, stomach and duodenum, a single instrument for the examination of upper gastrointestinal tract is available now. Here we report our experience with 112 cases examined by the forward-viewing esophagogastroduodenoscope (Panendoscope), manufactured by American Cystoscope Makers Inc. (A.C.M.I.).

Materials and Methods

All of the 112 patients on whom 115 examinations were performed were inpatients at VA Hospital, Topeka, at the time of examination. Represented were 110 males and 2 females. Their ages ranged from 24 to 82 years; 9 patients were past age 75. *Table 1* shows the indications for endoscopy. The patients were kept fasting for 12 hours prior to procedure; continuous nasogastric suction was performed for four hours before the procedure. Ice water lavage for a few hours was done before endoscopy in cases of active upper gastrointestinal bleeding. Demerol 75 mg and atropine 0.4 mg were given intramuscularly one-half hour before the procedure. Topical anesthesia of the fauces and posterior pharyngeal wall was obtained by use of 2 per cent tetracaine.

TABLE 1

INDICATIONS FOR ENDOSCOPY
115 Examinations

Epigastric pain	42
Upper gastrointestinal bleeding	37
Heartburn	12
Anemia and weight loss	6
Nausea and vomiting	10
Dysphagia	8

The ACMI-Lo Presti esophagogastroduodenoscope, model FO-7089A, which has a working length of 105 cm, was used. It has a 70-degree field of view and facility for distal tip manipulation of 120 degrees in two directions. The light source used was FCB 1002 for both routine examination and still photography. A 35 mm Olympus Pen FT still camera with a 70 mm Zuiko lens was used; cine photography was not used. The camera was attached to the fiberscope by means of a Lipmann camera adaptor. Wherever indicated, a biopsy of the lesion was obtained by the biopsy forceps supplied by the manufacturer along with the fiberscope.

The lower end of the fiberscope was lubricated generously with 2 per cent viscous Xylocaine. The fiberscope was passed with the patient in a sitting position and facing the examiner. After the gastroesophageal junction was negotiated, the patient was put in a supine position and the rest of the examination was completed in that position. A thorough examination of the whole stomach was performed before attempting an examination of duodenal bulb and postbulbar area. The pylorus was negotiated by maneuvering the tip as close as possible to the pyloric outlet and then applying gentle pressure to enter the duodenal bulb. The superior duodenal fold was identified and the rest of the bulb was examined by distal tip manipulation. After examination of the bulb, the instrument was advanced further to visualize the second part of the duodenum. No attempt was made to visualize the pylorus from the duodenal bulb by inversion technique described by some workers.[2] The esophagus was more thoroughly examined while withdrawing the fiberscope from the stomach into the esophagus. The gastroesophageal junction was regularly examined by inflating the esophagus. Constriction of the diaphragmatic hiatus could be identified. When a hiatus hernia was present, the supradiaphragmatic part of the stomach could be identified in many cases. For re-

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moval of mucus and occasional cleaning of fluid from the stomach, the suction mechanism built into the light source was used. After the completion of the procedure, the patient was kept fasting for another three hours, to avoid aspiration as the cough reflex was depressed following the use of topical tetracaine for pharyngeal anesthesia.

Results

The fiberscope could not be passed on two occasions; once the patient was uncooperative, and on another occasion marked lordosis and spur formation in the cervical spine precluded safe passage of the instrument. The lesions found on fiberscopy are depicted in *Table 2*. Extensive use of photography was made for better evaluation of the lesions *(Figures 1-6)*. Extensive use of the biopsy forceps was also made for histological confirmation of the lesions. A duodenal examination was attempted on 105 occasions. It was successful on 93 occasions, with a success rate of 88 per cent. In all the patients in whom the duodenal bulb could be entered, it was possible to visualize the postbulbar area and part of the descending duodenum. The Vater's papilla was seen on three occasions; however, attempts at cannulation of the pancreato-choledochal ductal system were unsuccessful with this forward-viewing instrument.

TABLE 2

LESIONS SEEN ON FIBERSCOPY
113 Examinations

Esophagitis	13
Benign esophageal stricture	6
Carcinoma of esophagus	1
Esophageal varices	4
Acute erosive gastritis	14
Benign gastric ulcer	11
Gastric carcinoma	1
Atrophic gastritis	7
Hypertrophic gastritis	2
Anastomotic ulcer	3
Duodenal ulcer	24
Duodenitis due to L-Dopa	1
Scarred duodental bulb	3
Gastric and duodenal ulcer	7
Granulomatous esophagitis	1
Normal examination	15

Ten per cent of the patients complained of mild transient sore throat the next day after the procedure. There was no morbidity, no complications, and no mortality from the procedure.

Figure 1. Benign esophageal stricture. Note the narrowed esophageal lumen at four o'clock position. White shiny areas on the left are optical artefacts.

Figure 2. Malignant esophageal stricture due to carcinoma. Note the nodularity around the esophageal lumen.

Figure 3. Esophageal varices.

Figure 4. Benign gastric ulcer. Note the biopsy forceps at eleven o'clock position.

Discussion

Various types of esophagoscopes and gastroscopes have been in use for the evaluation of the upper gastrointestinal tract but, until a decade ago, endoscopy played only a minor role in the management of patients with gastric disease because satisfactory instruments were not available. The advent of flexible, fiberoptic instruments has made the procedure more useful and safe.

The special features of forward-viewing ACMI esophagogastroduodenoscope have allowed us a satisfactory visualization of the esophagus, stomach, duodenal bulb, and postbulbar area. The value of the procedure has been enhanced by facilities for photography of the lesions *(Figures 1-6)* and also by the ability to perform accurate target biopsy of the lesion *(Figure 4)*. The increased maneuverability of the tip with a bidirectional tip-controlling mechanism has facilitated the passage of the fiberscope into the duodenal bulb and even into the descending duodenum. Other workers[3] have reported a better success rate than our 88 per cent, however, they were using the narrower, more flexible side-viewing system. Examination of the duodenal bulb should be considered as part of a general survey of esophagus, stomach, and duodenum because suitable instruments are now available for this purpose.[4, 5] Despite strenuous efforts during the course of 1,000 examinations of the stomach with an older instrument, the authors were unable to examine the duodenum.[7] Although endoscopy and photography have greatly facilitated the diagnosis of gastric ulcers, the need for biopsy and cytology in certain cases cannot be denied and may permit a more logical plan for therapy.[8]

The visualization of the duodenal loop and the Vater's ampulla via the duodenoscope has encouraged some workers[9, 10] to cannulate the ampulla and perform pancreato-cholangiography to visualize the pancreato-choledochal ductal system and, thereby, help in the diagnosis and management of patients with diseases of the pancreas and hepatobiliary system.[11] In our series, we were able to visualize the ampulla on three occasions, but were unable to cannulate it with this instrument; a side-viewing fiberoptic system is probably mandatory for such cannulation.

The indications for gastroscopy are listed in *Table 1* and the lesions seen are listed in *Table 2*. Gastroscopy was found particularly useful, and should not be delayed, in the diagnosis and management of cases with upper gastrointestinal bleeding. This has been the experience of other workers also.[12, 13]

In our series, there were no complications and no mortality from the procedure, even though the patient population was relatively older (9 patients

Figure 5. Acute erosive gastritis.

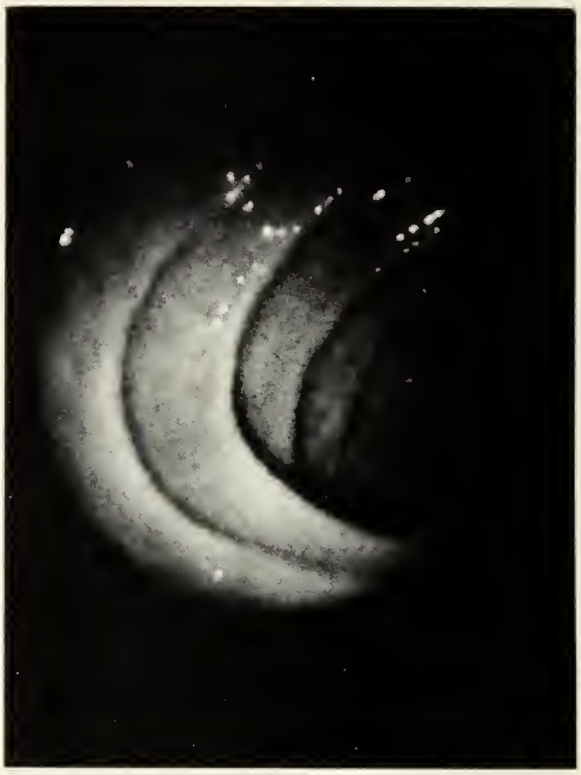

Figure 6. Normal duodenum; postbulbar region.

were past age 75) and many were psychiatric patients. In the reported literature, perforation represents the most serious complication of gastroscopy. The incidence of perforation of pharynx, esophagus, or stomach has been reported to be as high as 1.6 per cent.[6] The flexible fiberoptic gastroscopes are considered safe, but five cases of perforation of esophagus with the gastrocamera fiberscope have been reported[14] and authors recommend that uncooperative patients be excluded. This author feels gastroscopy can be safely carried out in agitated patients by proper psychological preparation, adequate premedication, and the use of local anesthesia. Cardiac arrest has been reported during endoscopy; this problem was not encountered in the present study. Deaths due to local anesthetics have been reported during gastroscopies,[15, 16] however, if the amount of tetracaine used is kept below 100 mg per examination, the danger is much reduced. Again, this complication was not encountered in the present series.

It is felt the fiberoptic esophagogastroduodenoscopy with attached photographic and biopsy facilities is a safe and useful procedure in the evaluation of patients with upper gastrointestinal problems.

Summary

Fiberoptic esophagogastroduodenoscopy was performed on 112 patients with various upper gastrointestinal problems. Photography and biopsy facilities were extensively used. Examination of the duodenal bulb and second part of the duodenum was successfully performed in 88 per cent of attempts. There were no complications and no mortality from the procedure.

Acknowledgements

The author is grateful to the nursing staff, Mr. B. G. McGinnis, and Mr. Delbert Rinehart for their help.

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(Continued on page 279)

Aortic Valve Replacement

Prosthesis Related Complications

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The surgical therapy for aortic valvular disease has improved markedly during the last decade. The operative mortality associated with replacement of the aortic valve has steadily decreased and there has been evolution of adequate valvular prostheses during this period of time.

THIRTY-SIX aortic valve prostheses were implanted in 35 patients at the Vanderbilt University Medical Center between April 1969 and June 1971. This series does not include patients in whom multiple valves were replaced. Patients who had concomitant procedures, other than replacement of another valve, were included. A disposable bubble oxygenator and hemodilution prime were utilized. In most instances, mild hypothermia (30-32C) and intermittent coronary artery perfusion were used during the period of valve replacement. Sixteen Starr-Edwards cloth-covered prostheses (series 2300, 2 patients; series 2310, 7 patients; and series 2320, 7 patients), 19 Bjork-Shiley prostheses, and one Wada-Cutter prosthesis were implanted. Patients were monitored in an intensive care area for the first few postoperative days, and anticoagulants were administered beginning on the second to fourth postoperative days.

Thirty-three patients were male, ranging from 8 to 72 years of age *(Figure 1)*. The cause of the disease, predominant hemodynamic lesion, and symptoms are tabulated in *Tables 1 and 2*. Patients with persistent class III or IV symptoms of pulmonary congestion were designated as having congestive heart failure. The operation was for replacement of a previously implanted malfunctioning prosthesis in four instances. Concomitant operative procedures were performed in eight patients *(Table 3)*. Segmental resection of the ascending aorta was accomplished in four instances for type II aortic dissections, and in one instance for a fusiform aneurysm. Two patients with aortic stenosis underwent simultaneous aortocoronary bypass for associated coronary artery occlusive disease, and one young adult underwent simultaneous closure of a congenital ventricular septal defect.

Results

Twenty-two severe complications were observed in 16 patients during the early postoperative period. These complications are listed in *Table 4*. There were only three postoperative complications which resulted in permanent deficits. In one patient, aphasia and right hemiparesis were noted on the night of operation. The neurologic deficit gradually cleared except for weakness of the right arm and hand. One patient experienced third-degree heart block at the time of valve implantation. The heart rate has been controlled by a permanently implanted pacemaker. The remaining patient, a 72-year-old man with associated atherosclerotic occlusive disease of the coronary and femoropopliteal arteries, developed an ischemic ulcer of the foot following a popliteal arterial embolectomy in the early postoperative period. The ischemic ulcer did not heal, and the patient had a below-the-knee amputation two months postoperatively. The remaining 19 complications cleared without residua. There were no operative deaths.

No patient has been lost to followup. The total followup represents 63.1 patient-years. Thirty-two patients have been followed for more than one year, and 17 for more than two years. Eight late major

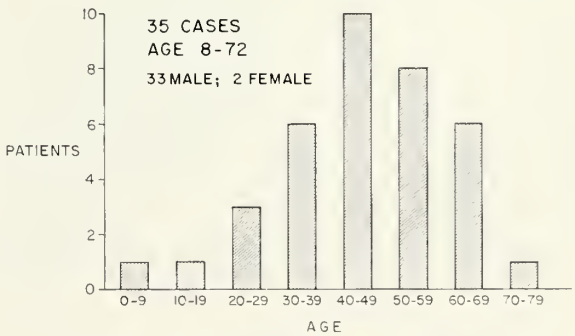

Figure 1. Distribution of patients by age.

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TABLE 1

ETIOLOGY AND PREDOMINANT HEMODYNAMIC LESION

Etiology	*Insufficiency*	*Stenosis*
Idiopathic calcification	—	13
Rheumatic heart disease	5	2
Dilatation aortic annulus	5	—
Aortic dissection	4	—
Congenital	3	—
Detached prosthesis	3	—
Hemolyzing prosthesis	—	1
Totals	20	16

TABLE 2

SYMPTOMS

Symptom	*Patients*
Congestive heart failure*	25
Angina or chest pain	23
Syncope	9
Myocardial infarction	1
Stroke (embolus)	1
Anemia (hemolytic)	1

* Class III or IV symptomatology.

complications occurred, three of these being fatal *(Table 5)*. One patient developed cardiogenic shock five months after aortic valve replacement and resection of the ascending aorta for type II dissecting aneurysm. At autopsy, a premortem clot was present on his Starr-Edwards prosthesis; no other possible etiology of the shock was evident. Another patient who had undergone resection of the ascending aorta and replacement of the aortic valve for acute dissecting aneurysm complained of pain in the neck. He died at home suddenly, 8½ months postoperatively. This patient had severe hypertensive cardiovascular disease and his hypertension had been controlled only with difficulty. It is thought that he experienced an extension of his dissection. No autopsy was obtained. The remaining complication was in a patient who was without symptoms until he died suddenly, 11 months postoperatively.

TABLE 3

CONCOMITANT OPERATIVE PROCEDURES

Procedure	*Patients*
Resection ascending aorta	5
Coronary artery bypass	2
Closure VSD	1

TABLE 4

POSTOPERATIVE COMPLICATIONS (30 DAYS)

Complication	*Patients*	*Residual Deficits*
Hemomediastinum	6	0
Severe arrhythmia	4	0
Neurologic deficits	2	1 (monoparesis)
Acute myocardial infarction	2	0
Wound infection	2	0
Heart block	1	1 (3° heart block)
Arterial embolus	1	1 (ischemic ulcer)
Ventilatory insufficiency	1	0
Wound dehiscence	1	0
Pulmonary embolus	1	0
Low cardiac output syndrome	1	0

His prosthesis was of the "close clearance" 2310 Starr-Edwards design. No autopsy was obtained. The incidence of prosthesis-related complications (both early and late) for each type of prosthesis implanted is tabulated in *Table 6*.

The rehabilitation of most patients has been gratifying. Except for the patient who suffered a late myocardial infarction, no survivor experienced severe cardiac disability. The usual response in patients with preoperative cardiomegaly is typified in *Figure 2*. Thirty of the 35 patients currently are pursuing vocations and hobbies commensurate with their age, and are not significantly handicapped by their heart disease.

Discussion

The operative mortality associated with replacement of the aortic valve has gradually decreased in recent years to a currently attainable level of 5 to 10 per cent.[1–3] There are also data available on the

TABLE 5

LATE COMPLICATIONS

Complication	*Time (Mos.)*	*Outcome*
Hepatitis	2	Recovery
Detached prosthesis	2½	Cure
Shock; clotted prosthesis	5	Death
Arterial embolus	6	Recovery
Myocardial infarction	8	Recovery
Aortic dissection	8½	Death
Endocarditis	10	Cure
Sudden death	11	Death

TABLE 6

PROSTHESIS RELATED COMPLICATIONS*

Prosthesis	*Followup*†	*Thromboembolism*	*Detachment*	*Endocarditis*	*Sudden Death*‡
Starr-Edwards	16 (26.8)	2§	0	1	1
Bjork-Shiley	19 (33.3)	1	1	0	0
Wada-Cutter	1 (3.0)	0	0	0	0

* Both early and late complications.
† Number of implants with number of patient-years in parentheses.
‡ Possibly related to "sticking" poppet.
§ Thrombus on prosthesis at necropsy in one patient.

late followup of patients with aortic valvular prostheses. In the Mayo Clinic series, approximately 70 per cent of all patients undergoing implantation were alive five years postoperatively. This survival is better than that documented for the natural course of aortic insufficiency or aortic stenosis, which is severe enough to produce certain ominous symptoms such as angina, syncope, and congestive heart failure.[1] It is encouraging that 91 per cent of the series of patients in the current report are alive with followups of up to three years.

The most frequent severe complication following aortic valve replacement is arterial embolism. The recent improvements in prosthetic valvular design have lowered the incidence of embolism to approximately five episodes per 100 patient-years.[4–5] Admittedly, the ideal prosthesis has not been developed; however, currently available prostheses afford results which are better than the natural course of most patients with untreated symptomatic aortic valvular disease.

Summary

Thirty-five consecutive patients underwent implantation of aortic valvular prostheses without operative mortality. There have been three deaths during an average followup of 1.8 years. Two of the

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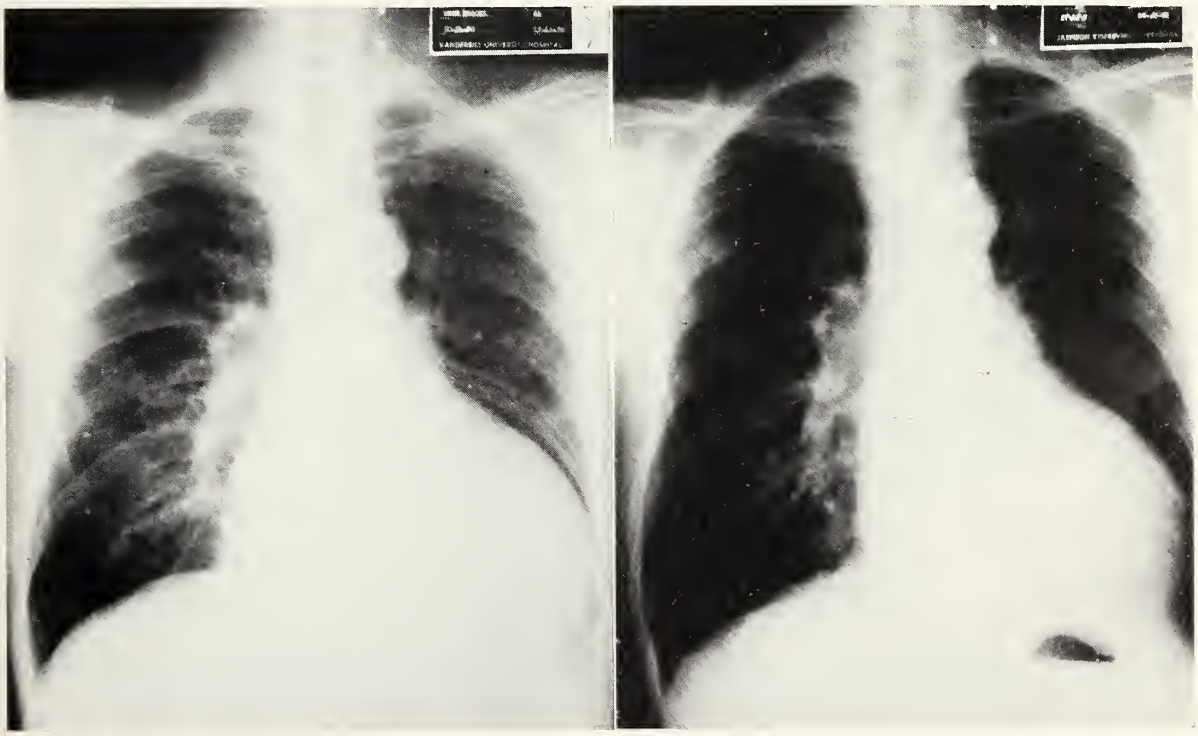

Figure 2. On left is preoperative chest radiogram of patient with aortic insufficiency; note marked cardiomegaly and prominent pulmonary veins. On right is radiogram made six weeks postoperatively; there has been a reduction in heart size and in pulmonary vascularity.

Source and Significance

Midsystolic Clicks and Late Systolic Murmurs

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SYSTOLIC CLICKS are extra sounds occurring in mid or late systole which are characterized by a sharp "snapping" quality. These clicks are usually heard best between the lower end of the sternum and the apex, and are usually sufficiently removed from the first and second heart sounds to distinguish them from split heart sounds. Midsystolic clicks were recognized in the last century, and the first contribution to their origin was made by Gallavardin who, in 1913, described necropsy findings of pericardial adhesions in three patients with midsystolic clicks. He ascribed the clicks to pleuropericardial adhesions, and this belief in the extracardiac origin of systolic clicks persisted in the literature for the next fifty years. In 1957, McKusick reported "that a late systolic click may occur on the basis of pleuropericardial adhesions (which) is suggested by its occurrence following clinical acute pericarditis, of rheumatic origin, for example." He further stated that the only clinical importance of the click was its timing in relation to the second sound in that the click -S_2 sequence, which carried a benign prognosis, could be easily mistaken for the S_2- opening snap sequence of mitral stenosis. Additional evidence for the extracardiac origin of midsystolic clicks was the variability of their intensity with positioning and respiration and the lability of their timing. Furthermore, in addition to their occurrence as a residual of pericarditis, midsystolic clicks have also been heard in patients with either mediastinal emphysema or pneumothorax.

In 1958, McKusick acknowledged the frequent existence of a short, sometimes musical, apical late systolic murmur which was initiated by a systolic click. He argued that this type of murmur, whether noisy or musical, was produced by rubbing of the pericardial surfaces, thus emphasizing its extracardiac origin. In 1961, Reid proposed that both the midsystolic click and the late systolic murmur were of mitral valvular origin: the click being due to abrupt tautening of the mitral chordae tendineae in midsystole, and the murmur being due to mitral regurgitation occurring in late systole. He speculated that redundant chordae or inadequate papillary muscle contraction could explain both phenomena, and the term "chordal snap" was proposed for the midsystolic click. Subsequently, a great amount of evidence accumulated which favored an intracardiac origin for the midsystolic click. W. P. Harvey devoted 18 years to the study of patients with midsystolic clicks in order to confirm the association between these clicks and pericarditis. In the great majority of the cases he studied, a complete cardiovascular evaluation, EKG, x-ray and special laboratory data failed to disclose evidence of pericardial disease. Therefore, his conclusion was that the midsystolic click was not usually related to pericardial abnormalities. These same observations were applied to the late systolic murmur, which was often associated with a midsystolic click. As was the case for the isolated midsystolic click, the late apical systolic murmur was not associated with pericardial disease.

Intracardiac phonocardiography was an important technique used to elucidate the origin of the midsystolic click and late systolic murmur. Ronan and Harvey, using this technique in 1965, were able to detect the existence of the late systolic murmur within the left atrium. However, upon making recordings on the right side of the atrial septum, the murmur promptly disappeared. Furthermore, the recorded intensity of the murmur increased following an Aramine drip, and decreased following inhalation of amyl nitrite. This type of response to these specific pharmacologic agents was characteristic of the systolic murmur of mitral regurgitation. Moreover, the intracardiac localization of the midsystolic clicks was identical with the localization of the late systolic murmur; the click increased in intensity and was earlier in systole after inhalation of amyl nitrite. It was, therefore, suggested that both the midsystolic click and late systolic murmur originated at the mitral orifice. The absence of the clicks within the right atrium was not only consistent with this hypothesis but also added weight to the evidence that midsystolic clicks were not merely transmitted from extracardiac structures.

More direct evidence of the mitral origin of the late systolic murmur was provided by left ventricular cineangiocardiography, which demonstrated late mitral regurgitation in all those patients studied who had clinical evidence of a late apical systolic murmur in addition to a midsystolic click. No mitral regurgitation was noted in those patients having an

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isolated midsystolic click without evidence of a murmur. Left ventricular cineangiocardiography also demonstrated prolapse of the posterior leaflet of the mitral valve in all patients having clinically detectable midsystolic clicks. This prolapse occurred regardless of the existence of a late systolic murmur, thus suggesting valvular competence in spite of prolapse in those patients with isolated midsystolic clicks. Moreover, evidence has accumulated to demonstrate that prolapse of the posterior leaflet of the mitral valve can occur in the absence of either a midsystolic click or a late systolic murmur. However, Criley, in 1966, using left ventricular cineangiocardiography and a simultaneous electrocardiographic timing device, has demonstrated that the click in midsystole coincides with maximal billowing of the posterior leaflet. Therefore, the explanation for the production of the midsystolic click will most likely be derived from the functional anatomy of the mitral valve.

Unfortunately, the precise pathogenesis of the mitral valve lesion and the mode of production of the auscultatory features in patients with midsystolic clicks and late systolic murmurs are unknown due to inadequate accumulation of anatomical data. Mitral valve disease and associated insufficiency may be a specific primary manifestation of Marfan's syndrome. In patients with this syndrome, the mitral valve leaflets are thickened, redundant, and of sponge-rubber consistency. These changes are especially obvious in the posterior leaflet which histologically demonstrates marked myxomatous degeneration. Furthermore, while the valve surface is markedly increased, the diameter of the annulus does not increase proportionally. The segments of leaflet between sites of chordal insertion have a tendency to bulge toward the atrium, giving the impression of "hoodlike protrusions," which might prevent complete closure of the valve and result in insufficiency. Moreover, as a manifestation of the connective tissue weakness in Marfan's syndrome, the chordae tendineae are elongated, thin, and delicate. This may allow the posterior leaflet of the mitral valve to prolapse into the left atrium during ventricular systole, a frequent angiographic finding in patients with Marfan's syndrome. Similar gross and histological observations were made in patients having clinically detectible mitral regurgitation associated with cineangiocardiographic evidence of a prolapse of the posterior leaflet of the mitral valve, but none of the other typical features of Marfan's syndrome (high arched palate, skeletal deformity, displacement of the ocular lens) were noted. This has been referred to as the "floppy valve syndrome" and, according to Read, probably represents a "forme fruste" of Marfan's syndrome. Pomerance, in a study of 805 hearts, reported that myxomatous valvular changes were not rare and that they occurred in all age groups. The severity of these lesions varied from localized areas of degeneration to gross ectasia with prolapse of the voluminous cusps into the left atrium during ventricular systole. Attention was drawn to the fact that the mural (posterior) leaflet of the mitral valve showed the most striking changes. The cause of the myxomatous changes in patients with Marfan's syndrome is unknown. However, it seems unlikely that aging of the leaflets is the sole cause, since many of the patients were young. The most satisfactory explanation based on the currently available facts is that the myxomatous degeneration and thickening of the posterior leaflet of the mitral valve were simply a tissue reaction which is the end result of many etiological factors, including both congenital and aging changes as well as a variety of diseases of "intermediate ages."

The precise relationship between the previously described anatomical abnormalities of the mitral valve and the production of the midsystolic click and late systolic murmur is obscure. Hemodynamic explanations for the late systolic murmur would appear to be invalid since the pressure gradient between the left ventricle and the left atrium is decreasing during the late systole. Furthermore, while both the Valsalva maneuver and the inhalation of amyl nitrite cause the systolic click to decrease in intensity and move earlier in systole, phenylephrine causes no constant alteration in timing or intensity. This implies that alterations in pressure are not important but that ventricular end-diastolic volume is possibly the major factor influencing the timing and intensity of the clicks. Both Valsalva and amyl nitrite cause a decrease in left ventricular end-diastolic volume. Based on these observations and Criley's evidence that the midsystolic click coincides with the maximal billowing of the posterior leaflet of the mitral valve, Barlow, in 1968, postulated that the midsystolic click was due to uneven distribution of tension in the chordal mechanism and that one or more chordae are lengthened or are relatively longer than other chordae. The sudden tautening of these lengthened chordae during systole could produce the clicking sound. It then becomes obvious that the smaller ventricular end-diastolic volume induced by amyl nitrite and the Valsalva maneuver will cause the elongated chordae to become too long relative to the long axis of the chamber earlier in systole. Maximum prolapse of the valve and the associated click would thus occur earlier in systole.

Similarly, a late systolic murmur might occur secondary to the lengthened chordae. These long chordae would be stretched during systole as the left ventricle decreases in size. Eventually, a point would be reached at which time the valve leaflet attached to the lengthened chordae would prolapse

into the left atrium, thus allowing blood to regurgitate from the higher-pressured left ventricle. It has been shown that the posterior leaflet of the mitral valve is connected directly to the left ventricular wall by chordae tendineae of the third order, which function as supports for the posterior leaflet during ventricular systole. Elongation or rupture of these chordae allow the central portion of the leaflet to billow, while the same changes occurring in chordae of the first or second order which control the leaflet edges cause the valve to become completely incompetent. It is, therefore, reasonable to assume that the primary lesion, causing the midsystolic click and the late systolic murmur, is lengthening of the third order chordae tendineae attached to the posterior leaflet of the mitral valve. This lengthening causes increased strain to be thrown upon the first and second order chordae, and it is possible that it is only when these chordae are stretched that mitral regurgitation ensues.

Another interesting theory concerning the pathogenesis of the midsystolic clicks and late systolic murmurs has been provided by Grossman, who observed that in patients with these two clinical findings, angiocardiographic studies demonstrated that the inferior aspect of the left ventricle appeared to bulge into the ventricular chamber during systole, thus forming a convex ridge in the area of the posterior papillary muscle and partially dividing the cavity into posterior and anterior chambers. Grossman also postulated that with ventricular systole, the inferior portion of the left ventricle bulges into the ventricular cavity lifting the posterior papillary muscle and chordae with it, thus allowing the chordae to become slack and, in turn, permitting the posterior leaflet to balloon into the left atrium. By the time the chordae again become taut, the posterior leaflet would have passed the point of normal apposition with the anterior leaflet so that there is some regurgitation into the left atrium. The systolic click would then be caused by the sudden tautening of the slackened chordae. Why the ventricle in patients with midsystolic clicks contracts so as to partition the chamber in systole is not known.

In 1965, Hancock and Cohn reported the existence of a distinctive syndrome of clinical features found in patients with midsystolic clicks and late systolic murmurs. Palpitations, chest pain, dyspnea, and symptoms of apparent neuropsychiatric origin (psychotic manifestations, hyperventilation syndrome) were the most common symptoms among these patients. Women were more commonly affected than men. Cardiac arrhythmias sufficient to explain the palpitations were noted in many of the patients. The most frequent arrhythmias were extrasystoles, which were both ventricular and atrial in origin.

The chest pain was not typical of angina pectoris. Usually, it was vaguely described as a sharp pain over the entire anterior chest and not related to exertion. Coronary angiograms, when performed, revealed entirely normal coronary arteries. The pain which lasted many hours was often associated with fatigue and frequently appeared to be of chest wall origin. Dyspnea was usually a mild symptom and was not accompanied by orthopnea, paroxysmal nocturnal dyspnea, wheezing, or other signs of congestive heart failure.

Several of these patients suffered from Marfan's syndrome or Turner's syndrome. Apart from findings typical of these associated conditions and the clicks, murmurs and extrasystoles, the remainder of the physical examination was unremarkable.

Phonocardiography revealed clicks occurring in the middle to later third of systole, and late systolic murmurs at the apex which were crescendo up to or slightly through the aortic component of the second sound.

Left ventricular cineangiocardiography demonstrated late mitral regurgitation and prolapse of the posterior leaflet of the mitral valve.

There was a high incidence of electrocardiographic abnormalities in patients with this midsystolic click-late systolic murmur syndrome. Low, flat, or inverted T-waves, S-T segment elevation, and prominent U-waves were frequently present in the lateral precordial leads (V_5 and V_6), as well as leads II, III, and aVF. Furthermore, the Q-T interval was prolonged in a majority of the cases.

The cause of the chest pain and EKG changes are obscure. Coronary artery disease is not the cause of the chest pain in that patients with the midsystolic click-late systolic murmur syndrome invariably have normal coronary angiograms. Furthermore, the chest pain is not associated with T-wave inversions on the EKG.

The EKG changes characteristic of the midsystolic click-late systolic murmur syndrome are compatible with posteroinferior myocardial ischemia or infarction. Even if posterior myocardial damage is present, it is difficult to explain its relationship to the valvular abnormality. It is not clear why massive dilatation of the posterior leaflet of the mitral valve should result in or be associated with posteroinferior infarction. Barlow and Bosman suggest that the dilated posterior leaflet, even though inside the left atrial cavity, can distort the circumflex coronary artery as it runs behind the left atrium in the atrioventricular groove either by pressure or by movement of the mitral annulus. This distortion could then predispose to occlusion or thrombosis of this vessel and produce posterior myocardial ischemia. Barlow further maintains that posterior leaflet billowing is more pronounced in patients with the most

widespread EKG change. However, as Jerasaty points out, many patients afflicted with the midsystolic click-late systolic murmur syndrome have normal electrocardiograms. Also, Criley has shown that pronounced billowing can occur in the presence of a normal EKG.

It is also tempting to suggest that primary coronary artery disease would cause myocardial ischemia, functional lengthening of the chordae, and consequent mitral insufficiency. However, the occurrence of the unusual EKG pattern in children, and of normal coronary angiograms in the presence of abnormal EKGs, is evidence against this postulate.

Engle, on the basis of Grossman's description of abnormal left ventricular contraction in patients with midsystolic clicks and late systolic murmurs, postulated that T-wave changes in the electrocardiogram could be attributed to regional disturbances in coronary blood flow at the site of unusual contraction. However, this theory has yet to be documented.

The high incidence of ventricular extrasystoles in the midsystolic click-late systolic murmur syndrome might be related to the prolonged Q-T intervals, in that prolongation of repolarization is thought to enhance initiation of extrasystoles. Neuropsychiatric disturbances might be linked to arrhythmias through the medium of hyperventilation. The respiratory alkalosis induced would cause a decrease in the serum level of potassium, which would result in a further prolongation of repolarization (*i.e.*, Q-T interval of the electrocardiogram) and enhancement of ventricular arrhythmias.

Ballooning of the posterior leaflet of the mitral valve is generally accepted as the underlying abnormality in the midsystolic click-late systolic murmur syndrome. However, as previously discussed, the precise etiological factor or factors underlying this abnormality are not well defined. In fact, in the majority of cases a precise etiology is not obvious and the syndrome thus becomes "idiopathic."

The prognosis for patients with the midsystolic click-late systolic murmur syndrome is, in general, benign. However, the occurrence of sudden deaths due to arrhythmias has been reported. Thus, antiarrhythmic therapy is sometimes indicated, especially when the electrocardiogram shows premature ventricular contractions interrupting the T-wave. Furthermore, bacterial endocarditis has been reported to occur in patients with either an isolated midsystolic click or a midsystolic click in addition to a late systolic murmur. Therefore, prophylactic antibiotics should be prescribed accordingly.

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Gastroduodenal Visualization

(Continued from page 272)

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Aortic Valve Replacement

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deaths were in patients who had undergone concomitant resection of the ascending aorta for dissecting aneurysm. One patient undergoing isolated aortic valve implantation has succumbed during the followup period. Two patients have experienced arterial embolism (an incidence of 3:100 patient-years). It is concluded that aortic valvular replacement now permits an improved survival over the natural course of aortic valvular disease once certain ominous symptoms such as angina, syncope, or congestive heart failure appear.

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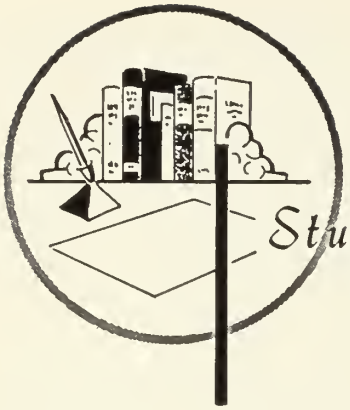

Student
THESIS

Pseudomonas Pneumonia

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When talking about pneumonia years ago, Osler[1] stated it was the "captain of the men of death." This was before our present antibiotic era which has, thankfully, demoted the captain to a sergeant. However, the physician must still use all the tools at his disposal to attack this disease and, far too frequently, he emerges the loser. With the advent of antimicrobials, many of the common etiologic agents of the past have been replaced by uncommon and, in many cases, pathogenically less virulent organisms. This paper will demonstrate, through a review of the literature, that Pseudomonas pneumonia is one of these disease processes. It will also explore the cause, pathology, and treatment of Pseudomonas pneumonia.

Of the numerous species of Pseudomonas, the one most often encountered in gram-negative pneumonia belongs to the species aeruginosa, previously known as Bacillus pyocyaneus.[2] For many years, the diagnostic bacteriologist considered all Escherichii and Pseudomonas found in feces natural nuisances which only hindered the search for Shigella or Salmonella.[3] This is not true any longer. Today, the bacteriologist is very much aware of the single, pair, or short chain of gram-negative rod noted in his microscope. *Pseudomonas aeruginosa* is usually motile (monotrichous) as well as Beta-hemolytic on blood agar.[2] These rods range between 1.5 to 3.0 x 0.5 meter in size and are nonsporing and nonacid fast. P. aeruginosa grows rapidly on usual media at an optimum temperature of 30C-37C, and 6.6-7.0 pH.

Since this organism is aerobic, it forms acid from glucose only, and produces no gas from fermentation. As mentioned earlier, P. aeruginosa was called Bacillus pyocyaneus, or the bacillus of blue-green pus, because it discolored its exudate with a pigment. In actuality, two pigments are produced: pycocyanin, a blue-green color, and fluorescin, a yellow-green.[3] This bacteria produces a proteolytic enzyme which enables it to hydrolyze gelatin, fibrin, casein, and albumin. This, along with the fact that it is usually localized in smaller blood vessels with consequent thrombosis and occlusion, easily explains why infarct-like areas of necrosis with a minimum of inflammation are frequently seen when infection results from this organism.

P. aeruginosa is found on normal skin, especially in the axillary and anogenital regions, and in 5 to 24 per cent of feces cultured.[4] It is notorious for the ease with which it colonizes liquids such as distilled water, optic medications, quartenary ammonia compounds, and soap solutions.[2] The usual sites of infection by this organism are the ear, genito-urinary tract, eye, respiratory tract, and blood stream.

In the preantibiotic era, gram-negative pneumonia was not of major concern to the physician. In 1947, the beginning of the antimicrobial era, Duerschner stated that the bacteria most frequently responsible for terminal bronchopneumonia were pneumococci, bacillus influenzae, beta-hemolytic streptococci and staphylococci. Rogers, in 1959, upon reevaluation, noted that pneumococci, streptococci, and bacillus influenzae had virtually disappeared as the major cause of mortality in patients suffering from pneumonia. He found staphylococcus infections were essentially unchanged since 1938. Present now, however, were the gram-negative bacillary infections. The significance of this was shown at autopsy where

*This is one of a group of theses written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Hirsch is now serving his internship at the Menorah Medical Center, Kansas City, Missouri.

11 per cent of autopsied patients were shown to have gram-negative pneumonia in 1959, as compared to 4.5 per cent in 1938.[5]

Yeh and his group evaluated 110 pneumonia patients from 1956 to 1963, and in 40 per cent of these patients Staphylococcus aureus was the cause of infection. Gram-negative microorganisms accounted for 27 per cent, and other gram-positive organisms were responsible for 23 per cent.[6] After evaluating his clinical records from 1938 to 1958, Rogers[7] found an increase in gram-negative pneumonia during the antimicrobial era from 1957-58, as compared to 1938-40 when the principal organisms were pneumococci, streptococci, tuberculosis, and staphylococcus. It was this author's contention that the significant etiological change he had observed was the result of the antimicrobial drugs effect on the usual floral pattern in these patients.

Kneeland[8] and his group also observed the same thing. They listed the microorganisms responsible for pneumonia in the antimicrobial era as: (1) staphylococcus; (2) P. aeruginosa; (3) Klebsiella aerogenes; (4) Escherichia coli; (5) alpha Streptococcus. In all cases of Pseudomonas pneumonia, the patients were on antibiotic therapy.

The increased incidence of Pseudomonas pneumonia cannot be denied. Certainly, the above data strongly, if not definitely, indicates that antimicrobial therapy is one of the major reasons for the change. However, antibiotics alone may not be of sole responsibility. The increased use of steroids in many patients with severe pulmonary disease must be considered. The gram-negative microorganisms become more prevalent when the normal bacterial flora is altered by natural or artificial methods, and when natural immunity is depressed.[9]

Sutler[10] studied this problem by isolating Pseudomonas from the respiratory tract of patients in a large hospital. He found that infected patients usually belonged to one of three groups: (1) elderly and debilitated individuals; (2) those on some type of antimicrobial therapy; and (3) those who had endotracheal suctioning or inhalation therapy. Considering the amount and extent of chronic obstructive lung disease seen in the elderly, it is not surprising that this group is included in his findings. The relationship between antibiotic therapy and gram-negative infections has already been discussed. The third group, those who had undergone endotracheal suctioning, has been examined by several investigators with some interesting conclusions.

Sutler himself found that three out of five patients with tracheotomies developed Pseudomonas infections. In 1965, Sanford's group thoroughly examined this problem. His figures showed that in 1952, at Parkland Memorial Hospital, the frequency of gram-negative bacillary pneumonia was 0.8 per cent. In 1963, the incidence was 8.0 per cent, or a 10 per cent increase. He felt the increased incidence corresponded to the increased use of inhalation therapy at this hospital. This was supported by the fact that the majority of inhalation therapy equipment incorporated reservoir nebulizers which were shown to generate aerosols containing large numbers of viable bacteria. These aerosols were of a size capable of penetrating beyond the level of the ciliated bronchial epithelium (1.4-3.5 n). Besides this fact, he noted that the aerosol's nebulizer jets were not being decontaminated by the standard cleaning technique. This resulted in the jets serving as a nidus for contaminating the reservoir fluid.

Sanford noted that the specific organisms isolated from aerosols varied from institution to institution. The major contaminants were, however, Pseudomonas species, Flavobacterium species, Herellea species, Alcaligenes species, and Achromobacter species. It was interesting that only gram-negative organisms were isolated. To further substantiate his claim, equipment tested without reservoir nebulizers did not show to generate aerosols containing numbers of bacteria in excess of that found in ambient air.[11]

Petty and Mitchell[12] agree that inhalation therapy contributes to the incidence of gram-negative pneumonia. They pointed out that when machines were not adequately sterilized between patients, a reservoir of gram-negative rods was dispensed to debilitated patients. They presented an interesting case of an 18-year-old college student who was hospitalized for treatment of an asthmatic condition. Apparently the patient was doing well until he was treated with an intermittent positive-pressure apparatus. Three days later he developed fever, chills, and cough along with purulent expectoration and hemoptysis. A diagnosis of necrotizing pneumonia and lung abscess was made by x-ray. P. aeruginosa was cultured. The authors strongly believe the inhalation therapy treatment was the source of this infection.

Cartwright[13] noted a thick "scale" on all surfaces covered with water in cape ventilators. Pseudomonas was cultured in every incidence. Grieble,[14] in further evaluating contamination in inhalation therapy equipment, showed that in a 20-bed respiratory disease intensive care ward, patients treated with intermittent positive pressure breathing therapy from the same machine developed one strain of Pseudomonas (Pyocin typing), rather than the many various strains usually seen in hospital settings. To further emphasize his point, this author showed that exclusion of humidifiers abolished gross environmental contamination by Pseudomonas and showed a marked decrease in sputum contamination.

To summarize, it appears certain that inhalation

therapy can be regarded as one of the main vectors for gram-negative bacillary organism, and especially Pseudomonas infection of the respiratory tract. Thus it is obvious that certain precautions must be observed to prevent continued infection.

Grieble states that 0.25 per cent acetic acid used for disinfecting purpose was unsatisfactory. Since tap water upon standing develops Pseudomonas contamination, it goes without saying that only sterilized saline or sterile water should be used in disinfected inhalation mechanisms, and certainly the aerosol mechanisms should be decontaminated between patients.

Along with inhalation therapy, Snow[15] has included tracheotomies as another source of gram-negative pulmonary infection. The factors he feels contribute to this include: (1) lack of filtration of inspired air usually performed by the nasal passage; (2) lack of humidification; (3) debilitated state of these patients; (4) trauma by the suction catheter; and (5) introduction of microorganisms via the catheter. The author feels this can be appropriately taken care of by using a soft, whistle-tip suction catheter and a Y-tube connector. Suctioning should only be done when the catheter is stationary in order to minimize mucus membrane trauma. Only sterile methods should be used, and benzalkonium chloride should be omitted as a catheter rinse and storage solution since it has been found to act as a mediator for Pseudomonas.

Duport in his study found that 80 per cent of his patients undergoing a tracheotomy procedure became infected with Pseudomonas. These findings were substantiated by Phillips and Spencer[9] in a series of eight patients suffering from chronic bronchitis who were treated with tracheotomy and mechanical ventilation. All eight patients subsequently became infected.

The morphological method by which the infection begins is still very much in doubt. Sprient[16] studied the nasopharyngeal flora in 53 adults and 84 children and concluded that there was present a large number of organisms capable of preventing growth of other types of organisms. Most of these were alpha-hemolytic streptococci, but a good number of gram-positive coccobacillus were also found. When these patients were given large doses of antibiotics (20 million units of penicillin and 12 gm of Dimocillin, along with one gram of streptomycin per week) this investigation found that 10 to 15 patients had undetectable nasopharyngeal colonies of streptococci and dramatic overgrowth of gram-negative organisms. Pseudomonas and Aerobacter made up 85 per cent of the population. The coliform preponderance persisted during therapy and lasted approximately six days after treatment. Even with the high doses of antibiotics there must be some organ or tissue response that allows these usually benign organisms to invade and infect.

Price[5] states that the occurrence of infection is determined by three variables: (1) virulence of the offending organism; (2) local or generalized host resistance; and (3) number and site of inoculation of organisms. Typically, gram-negative organisms are of a low order of virulence for pulmonary tissue and thus Price postulates that the other variables must contribute to gram-negative pulmonary infections. The fact that gram-negative infections are more prevalent in the aged and debilitated has already been discussed. Needless to say, malignancy, renal or cerebral disease, as well as congestive heart failure and preexisting pulmonary disease are adequate prerequisites for gram-negative pneumonias.

Sanford,[17] in studying the ability of the respiratory tract to clear P. aeruginosa organisms, noted that the clearance was largely dependent on the phagocytizing ability of the alveolar macrophages. He hypothesized that each bacterial species may have its own characteristic rate of clearance from normal lungs. This was shown to be the case when Sanford was able to identify three different patterns of pulmonary clearance of various pseudomonads. One strain of P. aeruginosa was capable of temporarily overcoming normal pulmonary defense mechanisms and multiplying in the lung during the first hour after aerosol deposition (Group I). Another strain was found to be destroyed by the normal defense mechanisms (Group III). The last group had an intermediate ability to withstand the defense mechanisms of the lungs and so were killed at a somewhat slower rate than the previous group (Group II). Upon examining these strains of Pseudomonas, Group I was found to produce greater amounts of extracellular lecithinase than the other two groups. The contention is that since pulmonary surfactant is made up of dipalmiloyl lecithin, Group I species may act in some way on the surfactant levels. This, however, does not answer all the questions and still more research needs to be done in this area.

Once the Pseudomonas organism is introduced into the lung tissue in a pathological state, certain microscopic changes occur in the tissue. Foley,[18] Price,[5] and Teplitz[4] described some of these microscopic changes. Proliferation of the P. aeruginosa occurs first at the capillary level with a resulting capillaritis. Once established in the capillaries, the organism proliferates along the adventitial channels to involve larger arteries and veins. It is believed that the resulting tissue necrosis can be attributed to the direct effects of bacterial proliferation along with the liberation of endotoxin and exotoxin. P. aeruginosa may also proliferate in the same manner around nonvascular structures, such as nerves or bronchioles. The microscopic lesions of gram-nega-

tive necrotizing pneumonia are those of alveolar septal necrosis.

Price states that traces of the preexisting alveolar septa are usually present and the alveolar spaces are filled with deeply basophilic, granular material containing large macrophage-like substance. These macrophage-like cells noted within the basophilic material have a clear zone or halo around them. In intimate association with these necrotizing lesions is involvement of the small vascular structures, as already described. The walls of these vessels, mainly small arteries, undergo necrosis with secondary thrombosis and arterial occlusion which produce small infarcts. Although speaking in general terms, Foley[18] emphasizes the presence of neutropenia. He suggests that the local suppression of neutrophils may be due to the soluble bacterial toxins. Since neutropenia is also present in sepsis, another theory is that there may be an arrest of maturation in the bone marrow resulting secondarily to circulatory bacterial toxins.

These observations certainly agree with those made by Hitchman and Kreibig in 1897, and Fraenkel[2] a few years later. Besides the pulmonary necrotizing aspect of this disease, Johnson[20] reported 129 cases of lung abscess caused by Pseudomonas organisms. These were felt to be mainly of embolic or thromboembolic origin.

The clinical picture of Pseudomonas pneumonia is similar to most of the gram-negative pneumonias. Dowling[12] described five usual clinical features: (1) it is usually not primary; (2) there is usually no blood or blood-tinged sputum; (3) it is not usually lobar; (4) there is usually no pleurisy; and (5) there may be variable leukocytosis. Tillotson[4] states that the Pseudomonas produce a lower-lobe bronchopneumonia frequently with large empyemas. In his opinion, this disease is more prevalent in the summer and autumn than at other times of the year. Lueberson[22] describes the clinical course of Pseudomonas pneumonia as including cyanosis, severe toxicity, apprehension, and confusion along with upper gastrointestinal bleeding. The Pseudomonas organism remains in the sputum long after the clinical signs and symptoms have cleared.

All investigators agreed that Pseudomonas pneumonia carried with it a very poor prognosis simply because it is prevalent in older debilitated patients who usually present with several significant underlying medical problems. Hinshaw[19] gives a mortality figure of approximately 50 per cent to the individual with a gram-negative pneumonia. This is certainly a depressing consideration.

The treatment of Pseudomonas pneumonia remains relatively unsuccessful even though antibacterial therapy in other infections has been remarkable. The reason for this, as previously mentioned, is that Pseudomonas pneumonia affects those patients suffering from other severe diseases, and those whose resistance to bacterial infection is permanently impaired. One can readily see that a bacteriocidal rather than a bacteriostatic agent must be used on the microbes at their site of invasion.

Vacek[21] maintains there are two reasons why antibiotic therapy has been unsuccessful. First, therapy is based on the theory that the antibiotic is efficient as determined by disc sensitivity. He feels this tells nothing of the bacteriocidal effects of a given antibiotic. Secondly, since most Pseudomonas infections are outside the vascular lumen, even in sepsis, the level of antibiotic may be too low for therapeutic results.

The main drugs used in the treatment of Pseudomonas infections are polymyxin B, colistin, gentamicin, carbenicillin, and various combinations of these. Polymyxin B sulfate is a basic polypeptide antibiotic which has been derived from various strains of spore-forming soil bacteria, Bacillus polymyxa. It has a bacteriocidal effect in vitro at concentrations of 0.5 to 20 units/ml against most gram-negative organisms including P. aeruginosa. Susceptible bacteria rarely acquire resistance to this antibiotic.[22] Ramirez[23] found most strains are susceptible to concentrations of 2 μg/ml. Since the drug is not readily diffusible across tissue membranes, its most therapeutic effect occurs when it is brought into direct contact with the susceptible organism at the site of multiplication. For this reason, plus the fact that when given parenterally in doses greater than 2.5 mg/kg, severe kidney irritation with the development of albuminuria and possible azotemia occurs, Ramirez experimented with endobronchial aerosol. In 72 patients, Lepper demonstrated that polymyxin B, administered in aerosol in doses up to 2.5 mg/kg/24 hrs, prevented implantation of P. aeruginosa in the trachea. Further investigation by Ramirez showed that 3.3 to 12 per cent of nebulized drug reaches the lung parenchyma. One patient was treated by using 300 mg polymyxin B in 10 cc normal saline aerosol three times daily for 3-5 minutes with the patient being rotated 30-45°. Possibly in the future more will be heard concerning this method.

Colistin is a complex polypeptide antibiotic derived from Bacillus polymyxa.[22] After deep intramuscular injection, this drug is rapidly absorbed, reaching peak blood levels within 1-2 hours and declining to trace levels in 12-24 hours. It is excreted in the urine. Clinically, colistin is bacteriocidal against most gram-negative organisms including P. aeruginosa. Unfortunately, the drug is extremely nephrotoxic and must be used with much caution in patients with renal disease. Urinary output, BUN, and serum creatinine must be evaluated often

if this drug is used. The usual dosage is 1.5-5 mg/kg/24 hrs in two to four divided doses.

Gentamicin is a broad spectrum antibiotic derived from the genus Micromonospora. It is an aminoglycoside closely related to neomycin and kanamycin. This drug is effective in vitro and in vivo against most microorganisms responsible for hospital associated infections, including P. aeruginosa, Klebsiella, Proteus, Escherichia coli, and Staphylococcus. The mechanism of action is believed to be interference with protein synthesis of the bacterial cell. Unfortunately, this drug, like the other two, is nephrotoxic as well as ototoxic. Holloway[24] studied 50 patients with bacterial infections treated with gentamicin. He evaluated urine output, hemogram, BUN, serum creatinine, serum bilirubin and serum glutamic oxalo-acetic-transaminase (SGOT), as well as audiometric testing before and after therapy. Twenty-nine patients had successful outcomes, with five qualified successes. He did not have any renal or optic problems but noted that serum levels of gentamicin were more closely related to urine output. He felt BUN and serum creatinine levels were not satisfactory guides and concluded that: (1) gentamicin was his drug of choice in Pseudomonas infections; (2) there was a low incidence of toxicity in his study; (3) dosage should depend on the severity of infection (5 mg/kg/24 hrs in very severe infections to be given in divided doses).

Holloway showed that in vitro the bacteriostatic range of gentamicin was 0.19-1.56 μg/ml, and the bacteriocidal range was 0.39-3.12 μg/ml. Darrell[26] in his study demonstrated that the minimal inhibitory concentration (MIC) of gentamicin in 45 fresh isolates was 4 mg/ml (20 strains). A 40 mg intramuscular injection twice daily produces a 2 μg/ml serum level after 3 hours. A 80 mg injection gives a peak level of 5.4 μg. Thus, in a 70 kg man, if a 5 mg/kg/24 hrs, given in divided doses, was used, 175 mg twice daily or 80 mg every 6 hours could be given to get bacteriocidal levels. However, this dosage could not be given to patients with renal difficulty.

Carbenicillin is a new semisynthetic penicillin prepared by adding a carboxyl group to the basic penicillin nucleus.[25] Since it is not absorbed when taken orally, it must be administered intramuscularly or intravenously. Most of this drug is excreted unchanged in the urine. Carbenicillin has been found to have significant activity in vitro against P. aeruginosa along with other gram-negative organisms. Chadwick's study showed that carbenicillin exerted a significant antibiotic effect against most of the 200 strains of Pseudomonas he used. Nearly all strains were inhibited by concentrations of 150 μg/ml. Five strains were resistant at this level, and this was not due to destruction by penicillinase-like enzymes to which the antibiotic is susceptible.

Chadwick found that a one-gram intramuscular dose of carbenicillin produced serum concentration of 14-48 μg/ml (the higher level with the aid of probenecid). Intravenous carbenicillin (1 gm) produced peak levels around 100 μg/ml. Both of these levels are ineffective for treatment of parenchymal infections. Since this drug has very minimal toxic effects (nausea, vomiting, increased SGOT, SGPT, and hypersensitivity), doses up to 40 gm daily may be given.[26] Jordon[28] reports an increased number of resistant strains of Pseudomonas to carbenicillin in Great Britain, where this drug is used extensively. Black[29] reports that he isolated six of ten strains with MIC greater than 5000/μg/ml. For this reason, carbenicillin used alone does not appear to be the answer.

Eickhoff showed that antibacterial synergism results when carbenicillin and gentamicin are combined, and antagonism results when carbenicillin and polymyxin B are used together.[31] In another study, Eickhoff had success in 25 of 42 patients treated with carbenicillin or carbenicillin and gentamicin.[30] Phair[32] demonstrated that by combining carbenicillin and gentamicin, bacteriocidal levels of 1.25 μg/ml of gentamicin and 25 μg/ml of carbenicillin were effective. This was felt to be due to the fact that carbenicillin induces spheroblasts which are inhibited by 0.625 μg/ml of gentamicin.

The conclusion to be drawn from these studies is that the combinations of these two drugs may make smaller therapeutic doses possible, with less chance of toxicity (ototoxicity and nephrotoxicity from gentamicin), and less chance of missing or causing resistant strains of Pseudomonas. Needless to say, this will not be effective in all cases and further investigation is still necessary.

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(Continued on page 291)

The President's Message

Do you as an individual practicing physician in the state of Kansas feel that it is important that the information which you obtain from your patient is treated in a confidential nature? I heard a phrase used the other day of "Computerization without careful control of thinking," and the concept that we have of confidentiality was brought to my mind. Our experiments with problem-oriented records, standardization of hospital records, and uniformity of other types of medical practice erode the concept of confidentiality.

Histories done in hospitals will make it considerably easier for the physician to tend to his handiwork in filling out the papers and, depending upon who asks the questions, how they are asked, and how the patient interprets them, you may get the proper answer for the question. This type of approach is also considerably easier to computerize than the one-on-one relationship which the physician develops with his patient.

Universal claims for third-party payors have already been computerized along with centralized computerization of forms which are sent in when a person applies for life insurance.

We will be placed under various types of rules and regulations, as a result of laws which are passed by our Congress and by our state legislature, to divulge more and more information about our particular patients. Computerization is a tremendous tool if used properly and if used in a proper setting. I think it is up to the physician to determine how the confidentiality of the information which he receives on a day-by-day basis from his patients should be utilized.

Who puts the information into the machine?

Thomas F. Taylor

President

Whose Truth Goes Marching On?

At the time of writing, the recent unpleasantness deriving from the activities in the well-known apartment-office complex shows no sign of prompt resolution. In fact, only two things seem certain. First, it will be the inspiration for study, evaluation, comment, and analysis for years to come. Second, no final assessment or disposition will be made that is satisfactory to all. Both of these observations stem from the fact that the loudly stated and oft-repeated objective is to determine the truth, and this is already an impossibility. We designate this goal as impossible not through any exhausted cynicism (which would be thoroughly justifiable at this point), nor yet through any grieved disillusionment, nor even any loss of faith in the democratic system. The impossibility lies in the fact that the elements have been so hammered and twisted that they have crystallized and shattered into a hopeless number of fragments—which is often the case in the search for truth.

With rare exceptions, each of the actors in this political morality play has protested that he spoke the truth at all times, and we accept this in memory of the numerous misbehaviors of our youth when, confronted by Maternal Authority and Incontestable Evidence, we stoutly and tearfully denied or defended the act and, in that moment of reckoning, were absolutely convinced of the truth of our interpretation. How, then, if all speak and act the truth, can things go awry when all of our numerous moral philosophies assure us that truth is the one final certainty, the indestructible defense against the forces of evil?

Truth is usually defined and used in the lexicon of any given group for its own purposes. It is an illusory concept at best. It suggests the Invisible Man of fiction who is rendered visible only by wrapping him in some material and assuming that the surface covering so produced (and of our own selection) accurately reproduces the inner character. It is exemplified by what we are pleased to call our Hearing Aid Principle. Anyone who is electronically equipped knows that sounds are of three types: (1) those we hear but no one else does; (2) those we cannot hear but everyone else does; and (3) those we hear perfectly well but no one else seems to have heard the same way. In each case, we are the ones who know the real truth and don't try to tell us otherwise or we'll get paranoid on you.

Depending upon your choice of emphasis, truth verges on wishful thinking or thoughtful wishing. Whether it is applied to electing a president or checking the family budget to see if a new outboard motor is possible, one tends to assume the necessity and then work backward to develop the truthful steps that will reach the desired end. Thus, the *modus operandi* can be justified at any point because it obviously represents the truth. The desirability of the result is the measure by which the validity of the process is determined.

What has this to do with the practice of medicine? Well, physicians are, by their own determination, the most truthful of men. They function only from truth. Diagnosis without truth is inaccurate. Therapy without truth is ineffective. Since medical care is the most personal of services, it follows that the physician is privy to the essential truths of mankind to a degree unequalled by any other social or intellectual discipline. He cannot be faulted for holding to this attitude in relation to the individual patient, but if one takes individual fragments of truth and puts them together, one does not necessarily build a total truth. The physician is not disposed to accept that anyone else is in as good a position to judge the truth about the patient as he is, but to assume that this gives him sole and unequaled capacity to establish what is good and truthful for mankind as a whole can be a dangerous self-deception.

Physicians, confronted with excessive criticism of their function, point out that they are being blamed for social and political ills not of their making. But if this is true (and it is), they can hardly be surprised that sociologists and politicians are forcing the issue of change. Physicians must perform in the area for which they are best equipped: advice and direction

toward patient care and the elements relating to direct medical service. If the changing structure reflects the influence of other disciplines, he must take care that he does not claim an expertise in these related areas while criticizing others for invading the purely medical area.

Individual physicians can at times be as contrary as a coat hanger in a crowded closet but they have one dangerous (though not exclusively medical) tendency: they love to talk to their colleagues who agree with them. The more they indulge in this comforting but myopic pastime, the more they equate their attitudes with inviolable truth, and anyone who doubts or questions assumes the status of a threat. Unfortunately, this prompts a defensive reaction which demeans, in the eyes of the layman, the value of any contribution they might make.

What are the two general comments most frequently made about the medical profession? First, the individual physician is, more often than not, considered a good man trying to do a good job. Second, organized medicine is a monopolistic, insensitive conglomerate intent on its own aggrandizement at the expense of the public. Neither of these assessments is, of course, totally true. Physicians are, for the most part, good men trying to do a good job, but they not infrequently err or fail or fall short of what they want to be for the same reasons other men fall short of their goals: they are human. Organized medicine *has* become a ponderous structure with organizational and bureaucratic faults common to any agglomerate, but along the way it has produced numerous benefits to physician and patient alike—now easily overlooked because they have been absorbed into the success of medical function.

Truth is identified with security. Once a concept of truth is established to fit a given purpose, any effort to question it or displace it is not interpreted as another noble contribution toward the definition of the fundamental principle of life. Instead, it is considered an assault, a threat to the bastion of truth which has been erected against the invaders, doubt and fear.

However remote the political fiasco may seem from the practice of medicine, then, it may serve as a reminder that isolation, introversion, and in-breeding of thought leads not to the strengthening of truth but a prostitution of truth. Then comes the misuse of a power that is strong and good until it is put to a false purpose.—*D.E.G.*

VOX DOX

Vox Dox Editor:

I am editing a book on renowned and notable physicians and their faith.

I am interested in obtaining contributors who have a special knowledge of the faith and/or religion of one or more notable and outstanding physicians. I am considering such physicians as Sir William Osler and Sir William Fleming, however, the notable physicians could still be alive.

Anyone interested in this project or who would suggest renowned physicians to write about may contact me at the following address: Claude A. Frazier, M.D., 4-C Doctor's Park, Asheville, NC 28801.

Thanking you in advance, I remain

Sincerely,
CLAUDE A. FRAZIER, M.D.

115th Annual Session

KANSAS MEDICAL SOCIETY

May 5-8, 1974, Topeka

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2. **Medico-Legal Aspects of Trauma**
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KANSAS OBSTETRICAL AND GYNECOLOGICAL SOCIETY
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KANSAS SECTION ON EAR, NOSE AND THROAT

PROFESSIONAL DIET COURSE ON THE HYPERLIPOPROTEINEMIAS

A pilot class taught by Mrs. Clara May Evanhoe, R.D., was held in Topeka in April 1973, for professionals to update and integrate their understanding of the lipid profile with the drug therapy and the diet therapy for the five types of hyperlipoproteinemias. The class was open to physicians, biochemists, registered nurses (especially coronary care nurses), and registered dietitians. Similar classes will be available at no cost to Kansas physicians and other professionals by arrangement with Mr. Bill Stanley, Program Director, Kansas Heart Association.

The introduction was a review of the many factors believed to be a part of the coronary picture, and a handout from *Journal of the American Dietetic Association*, March 1973, traced atherosclerosis from birth through increasing involvement to death. The statement of the Council on Food and Nutrition 1972, of the American Medical Association, was basic as was the acceptance of the international classification of the hyperlipoproteinemias. Next came a plan for screening with plasma, cholesterol, triglyceride, and total lipids laboratory testing; a further screening with electrophoretic studies, ultracentrifugation, free fatty acid level, and lipoprotein lipase assay; drug or diet therapy advisability after cost of treatment and patient motivation considerations. The physiology of lipid absorption, transport, and metabolism were thoroughly reviewed. The question of genetics of the hyperlipidemias was covered and the percentages of each type of those with hyperlipoproteinemias were given. Types I and II may well concern the pediatrician, because symptoms of these lipid disorders appear in childhood. The lipid profile plasma appearance (refrigerated overnight after a 12-hour fast), cholesterol levels, and triglyceride levels were discussed for normal and each of the five types.

Examples of normal and abnormal electrophoretic studies were shown and blackboard drawings were made of normal lipid profiles and of each of the five types. Symptoms frequently found with each of the five types were differentiated.

The primary hyperlipoproteinemias were differentiated from the secondary ones, which are due to some underlying disease that needs treatment. Diseases to be excluded before classifying a patient as either Type I, II, III, IV, or V were listed for each type in the professional handout materials.

Drugs of choice for treatment of the five types of hyperlipoproteinemias were considered and a handout from *Annals of Internal Medicine* 77:267-94, 1972, was distributed. Type I has only diet therapy as no drug therapy is presently effective. Types II, III, IV, and V have several drugs for use. The chart referred to listed only nicotinic acid, cholestyramine, d-thyroxine, and clofibrate as to their effects on plasma lipoproteins, the initial dose, the maintenance dose, adverse effects, special cautions, and the lipid types for which that drug was effective.

All authors referred to in the course agree that diet and drug therapy together get better results than either alone. Type I is an exception and is treated by diet only, since no drug is effective for this condition. The Heart and Lung Institute, Bethesda, Maryland, has published a series of booklets for a patient with any one of the five types, and a sixth, larger booklet for physicians, which explains diets for all five types of hyperlipoproteinemias. These diets were the basis of class diet therapy. Menu ideas and suitable food products expanded the basic dietary therapy. Brand names and market sources were given. If special knowledge for diet preparation was needed for some type, this also was given, as for example how to use MCT or cocoanut oil for Type I very low "long-chain-fat" diet.

Since 40 per cent of the elevated lipid population are Type II, and 40 per cent are Type IV, special stress was given to diet for these two types.

The objective of the class was to bring together into one single focus interpreting laboratory lipid results, drug therapy and diet therapy, and to familiarize participants with where foods allowed could be procured. According to unanimous evaluation by the students, the help offered is practical and pertinent for professionals who deal with cardiovascular patients. Further information on such a seminar or class for your area of Kansas can be obtained from the instructor, Mrs. Clara M. Evanhoe, R.D., or from Bill Stanley, Kansas Heart Association, 5229 West 7th St., Topeka, Kansas 66606.

Pulmonary Nocardia Asteroides

(Continued from page 268)

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KMS Obituaries

HAROLD L. COLLINS, M.D.

Dr. Harold L. Collins, of Beloit, died May 6, 1973 at the age of 68. He was born June 16, 1904, in Parsons.

Dr. Collins was graduated from the University of Kansas School of Medicine in 1931. He opened his medical practice in Beloit in 1932.

Surviving Dr. Collins are his wife and two sons.

CLINTON C. FULLER, M.D.

Dr. Clinton C. Fuller, of Columbus, died March 15, 1973 at the age of 80. He was born June 20, 1892, in Gresham, Nebraska. Dr. Fuller was killed in a two-car accident.

Dr. Fuller was graduated from the University of Kansas School of Medicine in 1919. He had practiced medicine in Columbus since 1926.

Survivors include his wife, a daughter, and two sons.

GEORGE H. GRIEVE, M.D.

Dr. George H. Grieve, 89, of Turon, died March 11, 1973. He was born December 6, 1884, in Westphalia.

Dr. Grieve was graduated from the Hahnemann Medical College, Chicago, in 1909. He had practiced medicine in Turon for 60 years.

Surviving Dr. Grieve are his wife and a daughter.

C. HERBERT MUNGER, M.D.

Dr. C. Herbert Munger, 92, of Emporia, died May 5, 1973. He was born March 8, 1881, in Waukesha County, Wisconsin.

Dr. Munger was graduated from the National University of Arts and Sciences Medical Department, St. Louis, in 1906. He had served in Emporia as the county health officer since 1932.

Survivors include his wife and a daughter. Memorial contributions may be made to the Heart Fund.

AMA Committee on Health Care of the Poor

Progress Report

THE COMMITTEE on Health Care of the Poor has been a standing Committee of the Council on Medical Service since the AMA Board of Trustees discharged its Ad Hoc Committee on Health Care of the Poor. The Ad Hoc Committee's functions were referred in late 1970 to the Council because of the related areas of interest. The Committee on Health Care of the Poor has emphasized the concern of organized medicine for the health and health-related problems of the poor in both urban and rural areas. It has maintained a close relationship with the Council on Medical Service and its other committees and with the Council on Rural Health.

Since reconstitution as a permanent Committee, six meetings of the full Committee and one Executive Committee meeting have been held. The first two meetings were basically organizational. In these, the Committee reviewed the many prior activities of the AMA concerning health care of the poor, including the activities of the Ad Hoc Committee on Health Care of the Poor, programs of the various AMA councils and committees, and other related Association activities. Also at these early meetings a Committee statement of purpose was developed, and it was subsequently approved by the Council on Medical Service.

The Committee's mission, as contained in this statement of purpose, is: to mobilize and utilize the resources of the medical profession and the AMA to achieve available, accessible, and acceptable health care for the poor, recognizing that performance must be commensurate with promise. To carry out this mission, the Committee outlined nine general functions and developed a work program for its own use that includes several goals and planned activities. Some of these activities can be implemented and completed in a relatively short period while others will, of necessity, be long range and thus dependent on the success of the Committee's short term activities.

As advocates of the poor, the Committee has served as the AMA group to which organizations and agencies representing the poor and poor people themselves can directly express their views. The Committee recognized early that it would be necessary to conduct its meetings in or near poverty areas if it was to obtain firsthand information and was to provide poor people with direct access to the Committee. Through three regional meetings, the Committee has provided inner-city ghetto residents, Spanish-speaking migrant workers, and poor residents of Appalachia an opportunity to relate their concerns to the Committee. In addition to poor consumers, the Committee has met with knowledgeable individuals from a variety of agencies and professions representing the fields of medicine, allied health, education, social services, economics, transportation, employment and manpower, environmental health, and government at its regional meetings.

The Council on Medical Service and its committees have long recognized that improvements in health care of the poor are dependent upon meeting local needs by local initiative and action. For this reason, the Committee on Health Care of the Poor thought it essential that state and local medical societies appoint committees on health care of the poor or assign this function to an appropriate existing committee. The AMA House of Delegates adopted a resolution in 1971 urging the creation of state and local medical society committees on health care of the poor. Since then, 23 state and 29 local medical societies have reported the establishment of committees concerned with health care of the poor, and these committees are in the process of developing local programs to improve health care services. The Committee has maintained a close working relationship with state and county medical society committees in an effort to stimulate local action and programs for the poor.

The Committee has met with several chairmen of state and local society committees at its regional meetings and through this medium of exchange has gained valuable ideas and information. The recommendations and ideas received and the summary minutes of these meetings have been periodically distributed to all state and local societies having committees on health care of the poor. The Committee intends to continue to keep local societies aware of its activities and to provide them with information learned about the needs of the poor and about potential action programs for improving their health care.

As one aspect of creating a better understanding of the specific needs of the poor, the Committee on Health Care of the Poor and the AMA Communications Division have developed a half-hour film on the health plight of the migrant agricultural worker. This film, entitled "Instead of Ed Dorado" depicts the living conditions of the migrant worker and his health and health-related needs at his home base and at his temporary work locations. The film was shown on a closed-circuit television at the 1972 Annual Convention in San Francisco and has been distributed

to a number of educational television stations and local medical societies on request. This film is representative of the Committee's work in developing health education materials in the language of the poor and reflecting their life styles. Other audiovisual materials are in preparation and in the planning stages.

From its meetings and many contacts with health professionals, the poor, and their representatives, the Committee on Health Care of the Poor has gained a great deal of insight into the health and health-related problems of those living in poverty. The Committee's observations contained in the Appendix either re-emphasize earlier statements made by the AMA or give specific emphasis to particular situations and problems that the Committee firmly believes should be understood by health care professionals and others concerned with health care of the poor.

The Committee on Health Care of the Poor recognized that its observations will require further elaboration and examination and for that reason believes that it is not appropriate to make recommendations at this time. The Committee also recognized that the majority of the problems existing in rural and urban poverty areas are not solely medical problems but instead are health-related problems—unemployment, substandard housing, overcrowding, inadequate education, poor environmental conditions—and correction of these conditions lies almost entirely outside the realm of medicine.

The poor have been studied and re-studied and are tired of the rhetoric and false promises resulting from these studies. Irresponsible promises have been made to the poor, and, because most promises have not been kept, they have generated mistrust rather than understanding and faith. If little can be done to solve the problems that exist in poverty areas, the Committee on Health Care of the Poor firmly believes that it would be far more prudent to promise only what can be delivered.

It is recognized that there are rapidly changing social forces exerting a strong and dynamic influence on health care delivery. To meet these pressures, the Committee believes that it is essential to maintain and improve the pluralistic system of delivery and financing. It is equally essential that local health care delivery systems be developed to meet specific local needs. The only hope of success lies within the community itself. It is incumbent on health professionals and community residents to develop cooperatively adequate health services that are available, accessible, and acceptable to the poor.

The Committee firmly believes that until there is a total effort directed toward improvement of overall quality of life for those who are poor, the grim picture of poverty and the plight of persons affected by it will continue to exist. Over the years, physicians everywhere have given their time and services. Much has been done, but much remains to be done. The time for action is *now*.

The Committee strongly believes that one of the greatest contributions it can make toward improving the health care of the poor is to offer its support to pilot projects that appear to offer new approaches for providing health care and that also appear to be sound and economically feasible. The Committee, being aware of all the above problems, consequently has under consideration the feasibility of assisting in the implementation of pilot action programs for Appalachia, for migrant workers, and for inner-city people.—*(Copies of the complete report are available from the AMA.)*

Pseudomonas Pneumonia

(Continued from page 284)

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(Continued on page 295)

Personalities—IN KANSAS MEDICINE

Frank X. Lenski, Jr., Iola, attended the KU postgraduate seminar at Emporia.

E. C. Altenbernd, Overland Park, announced that the name of the Kansas Tuberculosis and Health Association has been changed to the Kansas Lung Association. The name change conforms with that of the National Tuberculosis and Respiratory Disease Association to the American Lung Association. Dr. Altenbernd is President of the Kansas chapter.

George Dyck and **Vernon Kliewer**, Newton, attended the American Psychiatric Association meeting in Hawaii.

Named the director of the Burnett Burn Center at KUMC was **David W. Robinson**, Kansas City.

Robert P. Sherman, Kansas City, is the new president of the Kansas Thoracic Society. **F. Calvin Bigler**, Garden City, was named president-elect, while **Daniel L. Scholzman**, Kansas City, was elected secretary-treasurer.

After contributing a total of 95 years to medical practice, brothers **Carl S. Newman** and **Clifford B. Newman**, Pittsburg, are retiring.

William R. Lentz, Topeka, participated in a workshop on arthritis.

W. P. Mazur, Osawatomie, participated in the 14th annual R.N. Day.

Max S. Allen, Kansas City, has been named the first recipient of the Hashinger Professorship in Gerontology at KUMC.

At the final meeting of the Books and People program at the Pratt Public Library, **William J. Porter**, Pratt, showed x-ray pictures in connection with a talk on the history of the American Indian.

William Nice, Topeka, presented a talk on the methadone treatment program to the Topeka Ministerial Association.

One of the attendees at the postgraduate medical study course on anesthesiology at KUMC was **M. Dale Atwood**, Kinsley.

John A. Lynch, Topeka, was elected vice president of the Kansas Chapter of the Arthritis Foundation.

Retiring after more than 40 years of active medical practice in the same community, is **Glenn H. Jackman**, Cimarron.

Karl A. Menninger, Topeka, has been honored with a mastership from the American College of Physicians. He was the second Kansas physician to receive a mastership from the ACP.

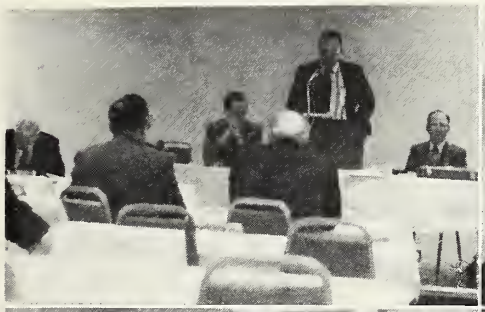
Speaking at a public meeting on the topic of cancer was **Renato B. Aves**, Parsons.

The Italians have put a clock on the Leaning Tower of Pisa. Reason: There's no use having the inclination if you don't have the time.

(Journal of the Mississippi State Medical Association)

Convention Glimpses—1973

(Courtesy of Chet Lessenden's and John Jarrott's cameras)



The D. C. Line

Editor's Note: The Editorial Board feels that the Kansas Medical Society would profit by maintaining contact with Representative Bill Roy, both as a source and interpreter of political activities on the national medical front. Accordingly, we are initiating a monthly column written by Dr. Roy. It is hoped that this will not only provide the service intended, but invite a response of readership either through the JOURNAL or to Dr. Roy directly.

This article is a continuation of the discussion I began last month in regard to the Canadian health care system.

Ninety-five per cent of physicians' payments in that country are fee-for-service. The fee schedule was agreed upon by the Canadian Medical Association and government. Median physician income net before taxes was $24,000 in 1966 (two years before Medicare), and $36,000 in 1970 (two years after Medicare). It is believed that present median income exceeds $40,000.

Family physicians were the lowest paid of all physician groups before Medicare. They immediately became and continue to be the highest paid. Over 50 per cent of Canadian physicians are family physicians, while in the U. S. only 20 per cent are so identified.

A notable redistribution of physicians occurred as soon as there was a universal payment mechanism. The redistribution resulted in practitioners locating where people were formerly unable to pay. For example, in three years the physician population ratio in Newfoundland went from 1:1600 to 1:1100. In addition, since the establishment of national health insurance, there has been a strong immigration of physicians. Today, 30 per cent of all physicians are foreign trained (in the U. S. 20 per cent are foreign trained).

Canada shares, with all other nations that I have visited, the problem of rapidly increasing expenditures for medical care. Expenditures are increasing at the rate of 12-13 per cent per annum. U. S. expenditures increased 10.6 per cent in 1972, despite Phase II controls. Present Canadian medical care expenditures are estimated at 7 per cent of the gross national product (GNP). U. S. expenditures are about 7.5 per cent of the GNP.

Canada is considering holding down the presently open-ended federal grants to the provinces by increasing the grants only by the percentage of increase in the GNP (9-10% rather than 12-13%). This appears to be an arbitrary decrease in the federal share. The federal answer is that they will also make available additional money to study and bring about changes in the health care delivery system. Prepaid group practice is one suggested change.

Good Physician Cooperation

I spoke at length with the Executive Secretary of the Canadian Medical Association, Dr. Bert Howard's counterpart. He indicated that there is presently high physician satisfaction with the Canadian system of national health insurance. Canada has built on a previously existing system and has preserved the voluntary hospital and private fee-for-service physician practice. They have also completely removed the barrier of cost for receiving medical care. Their system of national health insurance is highly decentralized.

I believe these are all worthy goals. While we cannot and should not wholly adopt the Canadian system here, we can learn from their successes and failures. All Canadians with whom I spoke believed that they had one essential ingredient in their modest success story, generally good physician cooperation and C.M.A. cooperation.—*W.R.R.*

PHYSICIANS IN POLITICS

Physicians have long been admonished to "get involved" in political affairs of this country. The results are becoming evident as more KMS members seek and attain offices at various levels of government.

The mayors of the largest and the fourth-largest cities in Kansas both are physicians: James M. Donnell, Wichita, and Jack D. Walker, Overland Park.

Three physicians serve as state legislators: John T. Brauchi, District 20; Leland Speer, District 37; Edward F. Steichen, District 119.

On the national level, William R. Roy serves as Congressman from the 2nd District.

Woman's Auxiliary

. . . There's no rest for the wicked, nor Auxiliary officers either

It's that time of the year when it's hard for Annie to get interested in anything but fishing, boating and all that fun stuff, but she'll try to give you a fast run-down on Auxiliary plans for the coming year.

Convention in Wichita was great, of course, with only a few snafus here and there . . . like the national officer who was to be the luncheon speaker getting sick and not able to come. Things went fine, though, as Mrs. Wendell F. Roller, our North-Central vice-president, came to the rescue and spent a couple of days with us. Since she was an expert at pinch-hitting, we presented her with a special baseball, bat, and pinch-hitter's hat.

In spite of the fact that most of the county Auxiliaries don't meet in the summer, much goes on at the state and national levels to get ready for the winter's activities. Our two vice-presidents, Mrs. Emerson Yoder and Mrs. Clair Cavanaugh, have already set up dates and places for the five regional workshops and are working out all the details. President Mrs. Lucien Pyle has mailed all her stationery (and that's more of a job than you'd think!) and is up to her ears absorbing knowledge about the Auxiliary as presented at the AMA in New York. It takes lots of doing.

Something else that's taking a lot of doing is the Health Education Conference Katie Pyle is planning for next year. A comprehensive, statewide meeting with all health agencies participating, this is to be another "first" for Kansas. It is to be modeled after the national conference held last year. Mrs. Dean Burnett is working with Dr. Kenneth Graham and Katie to put on a real show.

According to the sage advice of Annie's Mom, "there is no rest for the wicked." Annie doesn't think her state and national Auxiliary pals are wicked, but she's aware that there's no rest for them in summer either.

The national officers have announced their plans for the coming year in the following areas of concern: In the health education category mental health, aging and homebound, nutrition and ecology will be stressed. Health services projects will promote programs for children and youth, especially the GEMS babysitter project, block mothers, safety, and blood donorship.

The national Auxiliary suggests that mental health is a concern for all ages. A young child may be retarded or have emotional disorders. Adults may be alcoholics or have any number of emotional problems. Aging and homebound projects will be concerned with setting up day care centers for the elderly as well as getting proper nutrition for those who may not be able to get out and help themselves. Several Kansas cities have established "Meals on Wheels" or mobile meals projects in which our Auxiliary women assist in various ways by delivering food, keeping books, or assembling food trays.

Ecology is a concern of the entire world and Auxilians are urged to see that good food and habitat are provided along with preservation of our natural resources.

Child abuse and the battered child syndrome will receive attention, with new laws to back up efforts in correcting this problem. Proper care for children through GEMS and Block Mother programs are flourishing in several places in Kansas. Flammable fabrics, dangerous toys, and misuse of medicines and poisonous household substances will also be a part of the Health Services Committee. A "Safety on the Streets" program sponsored by the National Safety Council tells women how to protect themselves from attack. They teach a little judo and some "dirty fighting," so you fellows better be nice to your wives! Pedestrian safety and blood donor programs are also on the national agenda.

You can see there's really a lot everyone can do with such a wide scope of possibilities. And we won't forget our old favorites AMAERF, International Health Activities, and legislation either. So, if you have a special problem area you'd like us to work on, let us know and we'll try.

Like I said, "there's no rest for the wicked" or for state or national officers, or even for Auxiliary Annie, because like show business, the column must go on, summer or not.

But right now Annie's going to quit and go fishing. . . . See you.

Auxiliary Annie

(Continued from page 291)

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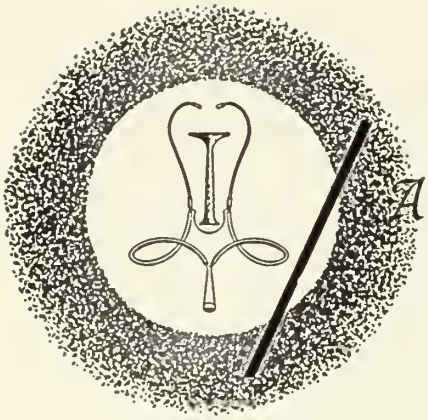

Announcements

Professional meetings, conferences, and postgraduate courses of national importance are listed for the* Doctor's Calendar. *Notice of the session is posted in advance to allow the physician time to make preparations.

JULY

July 14 — Thyroid Update Symposium, Broadmoor Hotel, Colorado Springs. Write: F. R. Gydesen, M.D., 2215 N. Cascade, Colorado Springs 80907.

July 22-27 — Flying Physicians Association, Sheraton-Harbor Island, San Diego. Write: Albert Carriere, 801 Green Bay Rd., Lake Bluff, Ill. 60044.

July 26-28 — American Gastroenterological Association. *Biliary Tract Disease.* Playhouse, Aspen, Colorado. Write: C. B. Slack, 6900 Grove Rd., Thorofare, N. J. 08086.

AUGUST

Aug. 19 — American Academy of Medical Administrators, 16th Annual. Continental Plaza, Chicago.

SEPTEMBER

Sep. 6-8 — American Association of Obstetricians and Gynecologists, Hot Springs, Va. Write: J. D. Woodruff, M.D., Johns Hopkins Hospital, Baltimore 21205.

Sep. 17-18 — AMA Congress on Occupational Health, 33rd Annual. Benjamin Franklin Hotel, Philadelphia.

Sep. 19-21 — Colorado Medical Society, Annual Meeting. Broadmoor Hotel, Colorado Springs.

Sep. 20 — American Society of Internal Medicine, Interim Meeting, Dallas. Write: W. R. Ramsey, 525 Hearst Bldg., San Francisco 94103.

Sep. 27-29 — Cancer of the Colon and Rectum, American Cancer Society 2nd National Conference. Americana Hotel, Bal Harbour, Florida. Write: S. L. Arje, M.D., 219 East 42nd, New York 10017.

Sept. 29 — College of American Pathologists & Bethesda Naval Hospital. *Sero-Diagnosis of Cancer Symposium.* Write: CAP, 1775 K St., N.W., Washington, D. C. 20006.

OCTOBER

Oct. 1-4 — Academy of Family Physicians, Denver. Write: Roger Tusken, Volker at Brookside, Kansas City, Missouri 64112.

Oct. 5-6 — Kansas Heart Association Annual Meeting, Hutchinson. Write: Bill Stanley, 5229 W. 7th, Topeka, Kansas 66606.

Oct. 15-19 — American College of Surgeons, Chicago. Write: C. R. Hanlon, M.D., 55 E. Erie, Chicago 60611.

Oct. 20-25 — American Academy of Pediatrics, Chicago. Write: R. G. Frazier, M.D., 1801 Hinman, Evanston, Illinois 60201.

Oct. 20-26 — Annual Otolaryngologic Assembly, University of Illinois Hospital. Write: Otolaryngology, PO Box 6998, Chicago 60680.

University of Colorado:

July 16-20	*Internal Medicine*
July 29	*Pediatrics*
Aug. 2-4	*Dermatology*
Aug. 16-17	*Gynecology*

For further information, write the Office of Postgraduate Medical Education, University of Colorado School of Medicine, 4200 E. Ninth Ave., Denver 80220.

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THE Journal OF THE Kansas Medical Society

AUGUST
1973

VOL. LXXIV
NO. VIII

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elp you determine initial dosage,
e possibility of side effects and
e ultimate prospects of success
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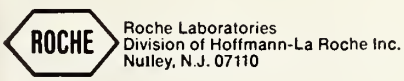

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Volume LXXIV, Number 8—August, 1973

The JOURNAL of the KANSAS MEDICAL SOCIETY

Contents for August

Scientific Articles

Myocardial Failure: Ventricular Aneurysm and Adynamic Areas—Franklin G. Bichlmeier, M.D., F.A.C.S., Thomas V. Thomas, M.D., F.A.C.S., William B. Toalson, M.D. and Sherman Steinzeig, M.D., Kansas City, Kansas . . . 297

Waardenburg's Syndrome: Identification of Patients—Ronald D. Greenwood, M.D., Boston, Massachusetts . . . 301

Clinical Cardiology

Computers and Cardiovascular Medicine—James W. Covell, M.D., La Jolla, California 303

Suppressed Plasma Renin Activity and Primary Aldosteronism—Paul I. Jagger, M.D., La Jolla, California . . . 304

Student Thesis

Actinic Keratosis: The Advent of Fluorouracil—Larry Anderson, M.D., Kansas City, Missouri . . . 306

Socio-Economics

Williams-Steiger Act: Occupational Safety and Health in Kansas—Lee Ellis, M.S. and Irvin G. Franzen, M.P.H., Topeka . . . 310

Miscellaneous

Advertisers . . . 18
AMA Committee on Community Health Care . . . 320
Announcements . . . 8
Book Reviews . . . 327
Classified Advertisements . . . 18
D. C. Line . . . 324
Editorial Comment . . . 317
Information for Authors . . . 18
Kansas Morbidity Incidence Report . . . 14
Medical-Legal Page . . . 321
New Members . . . 309
Obituaries . . . 319
Officers, Councilors . . . 328
Personalities in Medicine . . . 325
President's Message . . . 316
Vox Dox . . . 326
Woman's Auxiliary . . . 323

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Scientific ARTICLES

Myocardial Failure

Ventricular Aneurysm and Adynamic Areas

FRANKLIN G. BICHLMEIER, M.D., F.A.C.S., THOMAS V. THOMAS, M.D., F.A.C.S., WILLIAM B. TOALSON, M.D. *and* **SHERMAN STEINZEIG, M.D.,** *Kansas City, Kansas*

MYOCARDIAL FAILURE as a result of arteriosclerotic disease of the coronary arteries has been the number one killer in the Western Hemisphere. Despite the advances made in preventive, diagnostic and therapeutic endeavors, approximately 500,000 persons, many of them in their 40s or 50s, succumb to the sequelae of coronary artery disease. Pharmacologic agents and supportive measures have been the most widely used means of salvaging the victims of acute myocardial infarction and controlling the symptoms of the residual damage to the heart on those who survived the acute episodes. During the past three decades, desperate attempts have been made by various surgical procedures to restore blood flow to the myocardium, or to correct the complications of myocardial infarction. Many of the earlier revascularization procedures have gone into oblivion with the adaptation of autogenous saphenous vein bypass grafts beyond the site of obstruction. This procedure has been accepted generally as the only physiologically sound approach in the treatment of patients with surgically correctible lesions in any of the major branches of the coronaries.[1, 2] Revascularization of an ischemic myocardium before the patient has an infarction, and later replacement of it with scar tissue, offer the only hope for many of the patients with coronary artery disease to return to a normal, or nearly-normal life style.

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Bethany Medical Center, Kansas City, Kansas, has been a pioneer in the care of patients with coronary thrombosis and the associated complications. The foresight and ingenious efforts of Hughes W. Day, M.D., in setting up the first coronary care unit in the nation at Bethany Medical Center in 1962, has been accepted all over the country as a means of saving thousands of lives every year.[3] During the past two years, Bethany Medical Center also has developed an active cardiac surgical program for myocardial revascularization in patients with coronary artery disease, as well as for surgical correction of some of the fatal complications of myocardial infarction. Our experiences with the first 100 consecutive patients who underwent coronary artery bypass grafts have been without an operative death. Relief of symptoms

Ventricular aneurysms and large adynamic areas are frequent complications of myocardial infarctions resulting in full-thickness scarring of a portion of the left ventricular wall. Angiocardiography is the best way to demonstrate the size and site of such aneurysms, as well as to detect the presence of thrombi in it. Concomitant revascularization of the remaining myocardium is indicated when there are reconstructible lesions in the coronaries.

and hemodynamic improvement in most of the patients have been gratifying. There have been three late deaths among these patients. The majority of the patients had bypass grafts to two or three vessels. Frequently, revascularization procedures have been carried out in conjunction with resection of ventricular aneurysms and large adynamic areas, valve replacements, and pacemaker implantation on those with brady arrythmias. Surgery has proved to be preferred over other medical measures in the management of structural defects of myocardial infarction, such as ventricular aneurysm, akinesia of the ventricular wall, infarcted papillary muscle and dysfunction of the mitral valve apparatus, interventricular septal defects and heart block. Most of the victims of such structural damage of the myocardium were crippled from cardiac decompensation until surgical procedures were feasible.

Clinical Experience

Our clinical experience with ventricular aneurysms includes a total of 13 patients who underwent surgical treatment. All of the patients had disabling angina and shortness of breath. Four patients had cardiac decompensation intractable to medical treatment. Three patients were women, ten were men; their age ranged from 39 to 68 years. Six patients had organized mural thrombus in the aneurysm; two of them were treated previously for peripheral arterial embolism. One patient had intractable ventricular irritability and six episodes of ventricular fibrillation prior to surgery. Another patient had a large ventricular aneurysm, mural thrombus, and EKG changes suggestive of acute myocardial infarction *(Figures 1A and 1B)*. After emergency coronary arteriography this patient was operated upon and an embolus to the distal right coronary was extracted. Surgical intervention for the aneurysms was undertaken six weeks to 11 years after the patients' myocardial infarctions. All but one of the patients had their aneurysms in the anterolateral wall or apical portion of the ventricle. In our experience with the first 100 coronary artery bypass grafts, there were seven patients with small ventricular aneurysms on the inferior wall near the atrioventricular groove where no surgical repairs were made. Plain chest x-rays were not even suggestive of ventricular aneurysms in approximately one half of the patients *(Figures 1A and 1B)*.

Temporary cardiopulmonary bypass, using a disposable bubble oxygenator, was employed in every instance. Anoxic cardiac arrest by cross-clamping of the ascending aorta was used prior to manipulation or opening of the aneurysm. Three patients had resection of the ventricular aneurysm only, because of the lack of suitable vessels for bypass grafts. The average pump time was 18 minutes for these three

Figure 1A. The contour of the left heart border is not suggestive of a large aneurysm on the plain film.

patients, and on an average only 1.3 units of blood were used. Resection of the aneurysm with a single aorta-coronary bypass graft was carried out in two patients, and the cardiopulmonary bypass time averaged 48 minutes. Eight patients had two saphenous bypass grafts done at the time of resection of the aneurysm; cardiopulmonary bypass time averaged

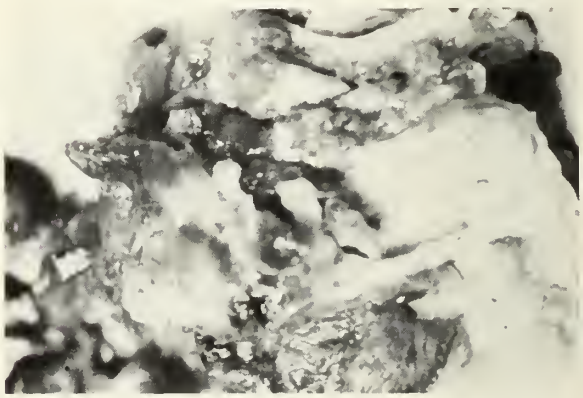

Figure 1B. Emergency left ventricular angiocardiography showed a large aneurysm and mural thrombus, despite the absence of an obvious aneurysm on plain film.

63 minutes, and the blood replacement was three units for most of them. While there were no operative deaths among the 13 patients, there was one late death in a patient who had extensive myocardial fibrosis and several episodes of ventricular fibrillation before surgical intervention.

When the ventricular aneurysm is of several years' duration and calcified, or when dense fibrous symphysis is present with the pericardium, this portion may be left on the mediastinal pleura to avoid injury to the left phrenic nerve. In other instances, the aneurysm is removed completely. Extreme care is taken to avoid injury to the papillary muscles or interventricular septum during the resection. Two layers of running synthetic sutures are used for closure of the defect after excision of the aneurysm *(Figure 2)*. The apical portion of the closure is used for placement of a ventricular sump before unclamping the ascending aorta.[4] The distal coronary anastomoses are done with the patient on cardiopulmonary bypass, while the proximal anastomoses to the ascending aorta are done after the patient is taken off the cardiopulmonary bypass. The relatively short pump run, in our experience, has been a factor in eliminating coagulation problems, renal failure, acute respiratory insufficiency, psychosis, and cerebrovascular accidents. Respiratory support was given to all the patients during the first few hours after surgery. Prolonged intubation of the trachea or tracheostomy was unnecessary in any of the patients. Eleven of the 13 patients had a postoperative hospital stay of ten days or less. Twelve of the 13 patients have been restored to a normal or nearly-normal level of physical activities for their age.

Pathophysiology

Ventricular aneurysms usually develop in an area of massive coronary thrombosis, which is transmural and heals with full-thickness scar tissue. Although the formation of aneurysms in the left ventricle and the presence of thrombus in it were described almost two centuries ago by John Hunter,[5] it was not until a century later that these were attributed to myocardial ischemia and coronary artery occlusion. Ventricular aneurysms usually develop in the anterolateral wall of the left ventricle and are the result of complete occlusion of the left anterior descending coronary artery or one of its major divisions. Szentpetery and associates[6] recently reported five ventricular aneurysms at unusual locations as a result of rare etiological factors. One of their patients was a child with a history of trauma to the coronary arteries, while another patient was a young woman with normal coronary arteries on angiography. Rosenthal *et al.*[7] recently reported the formation of aneurysms at the right ventricular outflow tracts in children who underwent total correction of tetralogy of Fallot. In their experience with 135 patients with outflow tract patches, eight patients developed definite aneurysms. Abrams and co-workers[8] estimated that 12-15 per cent of all patients with past myocardial infarctions would develop a ventricular aneurysm. Segmental areas of fibrosis in the left ventricular wall may produce uncoordinated contractions (dyskinesia), or complete absence of contractions (akinesia). An area lacking in effective contraction is just as devastating to ventricular function and efficiency as an area of paradoxical motion.[9] Lillihei and associates[10] feel that if more than 20 per cent of the cross-sectional area of the interior of left ventricle is involved by ineffective contractions, the patient will experience intractable, chronic heart failure.

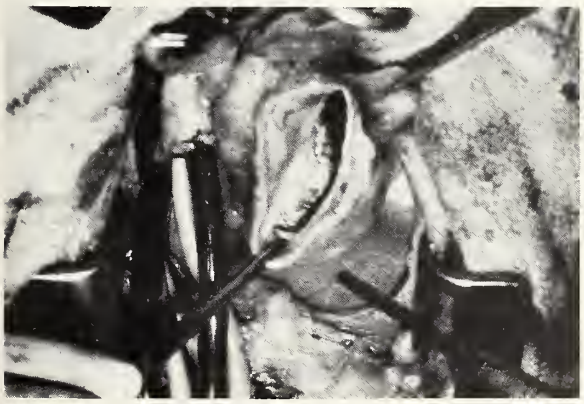

Figure 2. After the aneurysm is excised there is a clear fibrous rim for satisfactory closure of the ventricle.

When the patient has an adynamic area from the scarred left ventricular wall, the intraventricular systolic pressure stretches this area to produce a bulge during systole. The power of contraction generated in the normal left ventricular myocardium is dissipated over a large surface area and forces a portion of the left ventricular contents into the aneurysm. This ineffective expenditure of energy during systole is responsible for the early symptoms of angina and later heart failure.[11] When the area of paradoxical motion is large, as much as 75 per cent of the left ventricular volume may be sequestrated into the aneurysm. Thrombosis on the scarred endocardium is frequent, particularly when there is chronic heart failure and stagnation of flow *(Figures 3A and 3B)*. Favolaro and associates[12] found clots in 64 of their 130 patients with ventricular aneurysms, although histories of peripheral emboli were obtained only on eight patients. Although sudden rupture of an infarcted myocardium is a frequent cause of early death in patients with massive myocardial infarction, the

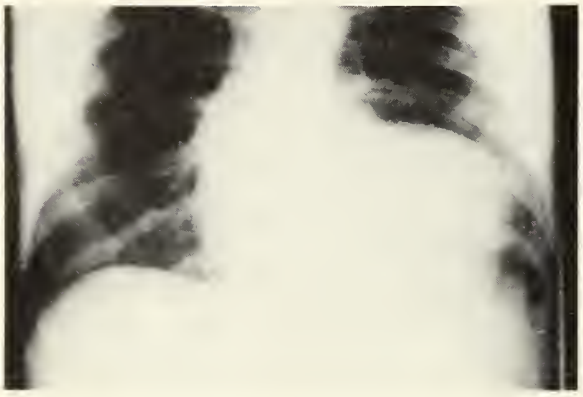

Figure 3A. Typical x-ray finding of a large ventricular aneurysm on plain chest film in one of the patients.

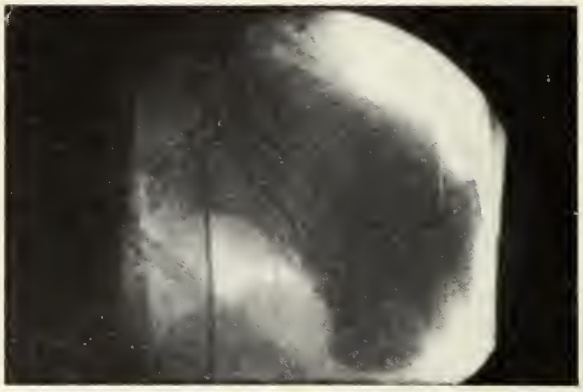

Figure 3B. Surgical specimen showing the fibrous wall of the aneurysm and large fragments of thrombus adherent to it.

rupture of a well-formed ventricular aneurysm is unheard of.

Diagnosis

Physical examination is usually unremarkable except when the aneurysm is large or when there may be a diffuse apical impulse. Until the development of cineangiocardiography, the diagnosis of ventricular aneurysms was made on chest x-rays. Although fluoroscopy may reveal large areas of paradoxical motion, it is an overrated step in the diagnosis of ventricular aneurysm. It is of interest in our series that sizable ventricular aneurysms showed no striking abnormalities on conventional roentgenographic studies. A systolic murmur may be present in some patients, although ventriculographic demonstration of mitral regurgitation is less frequent. When there is significant mitral regurgitation, it is usually the result of papillary muscle dysfunction. The papillary muscle dysfunction is usually responsible for an accentuated first heart sound in many patients with ventricular aneurysms. Electrocardiography is not particularly valuable in diagnosing ventricular aneurysms despite the presence of characteristic S-T segment elevations in some patients.

Various mediastinal and pericardial lesions may complicate the diagnosis of ventricular aneurysms on routine chest x-rays. Pericardial cysts or calcifications, localized effusions, redundant mediastinal fat pad, and vascular lesions of the thoracic aorta may pose diagnostic difficulties.[13-16] Left ventricular angiography is the most diagnostic step in confirming the presence of an aneurysm. Routine ventriculography with selective coronary arteriography has been helpful in delineating the site, size, presence of thrombus, and mitral regurgitation in patients with suspected or unsuspected ventricular aneurysms. In a report of cineventriculography in 100 patients with coronary artery disease, Cheng[17] found the presence of a ventricular aneurysm in 35. Only seven of these 35 patients showed any abnormality of the contour of the left ventricle on chest x-rays. Patients with ventricular aneurysms and large adynamic areas usually have an elevated end diastolic pressure.

Treatment

The first aneurysmectomy by a closed technique was reported in 1955 by Likoff and Bailey.[18] The technique of temporary cardiopulmonary bypass for resection, as adopted today, was first used successfully by Cooley[19] in 1958. The ideal time for surgical intervention in patients with symptomatic ventricular aneurysms is 10-12 weeks after a myocardial infarction. The operative risk for post-infarction ventricular aneurysms has been surprisingly low in our experience, particularly in a patient with far advanced coronary artery disease. Development of symptoms, such as angina, heart failure, irritability of the myocardium, significant mitral insufficiency, and peripheral embolizations, is a definite indication for surgical intervention in patients with a ventricular aneurysm. Direct myocardial revascularization in a majority of the patients, in addition to resection of the aneurysm, has been an important factor in minimizing postoperative cardiac problems and improving the patient's myocardial performance.

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(Continued on page 302)

Waardenburg's Syndrome

Identification of Patients

RONALD D. GREENWOOD, M.D.,* *Boston, Massachusetts*

WAARDENBURG first described the group of malformations which bear his name in 1951, in Holland.[1] The major abnormalities in children with this syndrome are lateral displacement of the median canthi, a broad and high nasal bridge, medial hyperplasia of the eyebrows, partial albinism, deafness, and an autosomal dominant pattern of transmission.[2]

The present case is reported to remind the physician of the manifestations of this unusual syndrome and the opportunities for identifying patients with this entity.

A child with Waardenburg's syndrome is described, and clues to early diagnosis are discussed.

Case Report

The child was born by a normal spontaneous vaginal delivery following a normal nine-month gestation to a 16-year-old mother and a 17-year-old father. Weight was 3.1 kg (6 lbs. 11 oz.), and length was 50 cm (19.5 in.). A white forelock was present at birth. The mother had one brother born prematurely and a sister who had petit mal epilepsy. Otherwise, the family history was normal. The father suffered partial deafness, a white forelock, and medial hyperplasia of the eyebrows. One of the father's brothers had a "reading defect"; otherwise, the paternal family history was normal. There was no deafness in the family on either side for two generations.

This patient was first seen at the Schilling US Army Health Clinic for a well baby examination at 18½ months of age, when the weight was 9 kg (third percentile) and length was 76 cm (tenth percentile), with a head circumference of 46 cm (less than fiftieth percentile). Examination *(Figure 1)* revealed lateral displacement of the medial canthi, a white forelock, marked medial hyperplasia of the eyebrows, a prominent and wide upper lip, flat nasal bridge, prominent hair growth onto the forehead, and irises of the same color. The infant was alert, happy, did not respond to loud noises, and could utter gutteral noises only. Other than the presence of a rhinitis, the remainder of the examination was within normal limits.

A Denver Developmental Screening Test (DDST) revealed performance compatible with chronologic age in gross motor, social and fine-motor adaptive areas, but grossly retarded in language. There was no response to a bell, no da-da or ma-ma, no imitative speech sounds, and no turning to voices; noises were uttered (laugh or squeal) but no specific repetitive vowels or other sounds could be discerned. An audiometric examination revealed a severe to profound hearing loss. A hearing aid was obtained and special training for both child and mother was initiated. Subsequent examinations revealed slow but improved language development. Genetic counseling was provided the parents.

Comment

This child displayed the typical findings of Waardenburg's syndrome. The most serious manifestation of this syndrome was the severe deafness. The patient's father also had this entity which was not previously identified because of the mildness of the condition.

This case serves to remind the physician of several possibilities for early identification. A history of deafness in the immediate family should be sought in any prenatal visit and the newborn carefully evaluated if such a history is present. If one of the parents exhibits a white forelock, suspicion should be aroused. A baby with a white forelock should be carefully evaluated for deafness on a regular basis from the initial examination in the nursery and throughout childhood. Since a significant number of patients with Waardenburg's syndrome arise by spontaneous mutation,[2] the absence of a positive family history should not deter the physician. Routine screening for deafness in the neonate and routine performance of audiograms in school are also useful procedures in any patient.

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The views of the author do not purport to reflect the position of the Department of the Army, or the Department of Defense.

Figure 1. Facial characteristics of Waardenburg's syndrome.

Waardenburg's syndrome is an uncommon disorder. Waardenburg estimated the incidence in Holland to be 1:42,000.[1] However, sufficient cases have been reported to warrant consideration of this syndrome by all physicians. Deafness is a major health problem in the United States, and this syndrome has outward manifestations which increase the likelihood of early diagnosis and treatment of the deafness.

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(Continued from page 300)

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(J. of the Mississippi State Medical Association)

"I think he's well enough to go home."

Clinical Cardiology

Computers and Cardiovascular Medicine

JAMES W. COVELL, M.D.,* *La Jolla, California*

Introduction

ALTHOUGH THE POTENTIAL of computing techniques in cardiovascular medicine has been recognized for some time, practical applications have become available only recently. Advances in technology coupled with rapid growth of a pool of individuals with special skills in biomedical computing have helped to propel computers to the forefront in cardiovascular diagnosis, patient care, and research at a cost that is no longer prohibitive.

There are two fundamental types of computers—analog and digital. An analog computer consists of linked networks of electronic components capable of analyzing and characterizing signals derived directly from physiological recordings. Thus, analog computers are used to analyze and filter the electrocardiogram; recognize and record the frequency of ventricular premature contractions; calculate and record cardiac output, heart rate, systolic, diastolic, and mean arterial pressures. These devices have been effective in intensive care and coronary care units. However, their use is limited since they possess no capacity for memory and are, thus, unable to correlate a patient's current physiological status with other variables, such as his past condition, previous history, or physical findings. Their effectiveness can be enhanced by combination with the second type of computer—the digital computer.

Digital computers are complex, high-speed, costly devices. They perform a wide variety of independent operations (additions, subtractions, multiplications, etc.) in microseconds; but they perform each operation one step at a time. Thus, they require a program that essentially "tells" the computer what step to take next and where, in the circuitry, to store resultant information. Accordingly, digital computing requires, first, the electronic hardware—a computer; and second, the software, *i.e.*, programs comprising instructions to the computer concerning a particular task. Both are equally essential to computer utilization.

Digital computation has been applied successfully to electrocardiographic diagnosis, patient monitoring, and the storage and retrieval of information derived from the patient's history and physical examination. Fortunately, existing hardware is now adequate for most tasks in these areas. Furthermore, numerous programs have been developed and tested, and are now commercially available.

Specific Applications

Electrocardiographic Diagnosis. During the past several years, programs have been developed for electrocardiographic interpretation utilizing signals obtained either from standard twelve-lead electrocardiographs or from vector cardiograph equipment. Naturally, some variation exists between a given physician's reading of an electrocardiogram and the computer's diagnostic analysis of the tracing. However, it is now generally accepted that computer analysis is effective in correctly classifying normal records and that good agreement between a human expert and the computer can be anticipated for most abnormal records as well. It is now possible to obtain electrocardiographic analysis by computer via ordinary telephone lines within minutes utilizing commercial services available in most areas. In some cases, computer recognition of abnormal tracings can be directly linked in order to initiate automatically additional expert consultation within a large center. Naturally, the computer cannot substitute for physician judgements based on the patient's previous electrocardiograms, history, or physical examination. In the assessment of these important diagnostic factors, as well as in the assessment of small variations in the electrocardiogram wave form, the computer cannot replace but can only supplement the skilled judgment of the physician.

Patient Monitoring. Perhaps the most rewarding and eventually most important application of computer electrocardiographic analysis will be in the area of intensive care monitoring. It is well recognized that prompt treatment of bradycardia or ectopic ventricular beats can lead to dramatic reductions in the mortality rate from acute myocardial infarction. Since programs are now available for on-line interpretation of arrhythmias and for on-line analysis of wave form, it is now possible to utilize computer diagnosis in the intensive care setting for on-line patient mon-

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itoring. Computer analysis of signals obtained with appropriate transducers can be used to monitor constantly not only the electrocardiogram, but also respiratory function, blood pressure, and other physiological functions. Programs have been devised which enable the computer to alert nurses or attending physicians whenever specified arrhythmias or alterations in hemodynamic or respiratory status occur. Computer systems for this purpose currently commercially available cost less than $10/day/bed and can significantly supplement conventional monitoring techniques.

Discreet Data Analysis. Another area in which computers now contribute effectively to cardiovascular medicine is that of maintaining medical records. Unfortunately, the extent of utilization of this important application has been disappointing. Theoretically, information entered in medical records can be classified and stored relatively easily with the aid of modern computing technology. Recent emphasis on the development of techniques necessary for direct computer-physician interaction and on utilization of problem oriented medical records may help to facilitate more widespread applications in this area. Computer-physician interaction can be utilized as follows: the computer is programmed to flash a question on an oscilloscope screen. The physician responds in a "yes" or "no" fashion, or by typing a response in ordinary English. The computer's questions continue, usually according to a "branching" logic pattern, similar to that employed by a physician obtaining a history. Thus, the computer might inquire whether chest pain had been present. An affirmative response would lead to additional questions concerning details including its radiation, intensity, duration, and frequency. Depending on the response, the computer would ask additional questions to obtain further relevant data. By this means, data can be obtained for storage and retrieval in a logical format. This approach has been applied to storage and retrieval of information in nurses notes, intake-output records, medication records, and physicians' history and physical examination reports.

Future Applications

Computing techniques will undoubtedly play an expanding role in cardiovascular medicine. Widespread utilization will probably occur first in the form of on-line monitoring in coronary care units and subsequently in electrocardiogram interpretation and large scale screening. Economic application of computing techniques to facilitate information storage and retrieval will ultimately be of considerable importance in effective maintenance and utilization of medical records for patient care as well as for investigation in cardiovascular medicine.

Suppressed Plasma Renin Activity and Primary Aldosteronism

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THE APPEARANCE in the 1960s of a relatively simple radioimmunoassay for plasma renin activity (PRA) has resulted in the rapid proliferation of literature relevant to renin changes in hypertension. At the same time, this radioimmunoassay has made the measurement of PRA available for general clinical use and, therefore, the physician who sees patients with hypertension needs to understand the significance of this recent literature in order to know when to utilize this tool. Renin, of course, is the enzyme produced in the cells of the juxtaglomerular apparatus of the kidney that acts on a circulating substrate to initiate the formation of angiotensin II. Angiotensin II is both a potent vasoconstrictor and a stimulus to aldosterone secretion by the adrenal cortex. The observation concerning renin that has received the most attention recently is the somewhat unexpected finding that as many as 25 per cent of patients with hypertension have suppressed peripheral PRA. This means that PRA remains low even after the patient has been subjected to a stimulus that would increase PRA in the normal individual. The standard stimulus usually employed involves placing the patient on a low sodium diet for 3-5 days and then having him ambulate for 2 hours. Suppressed PRA has been emphasized in the past as a critical finding in patients with primary aldosteronism. Though this is true, it now has been shown that the majority of patients with suppressed PRA do not have aldosterone hypersecretion and an alternate explanation for suppression is needed in these cases.

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Primary Aldosteronism

To review the situation in primary aldosteronism, autonomous hypersecretion of aldosterone by a lesion in the adrenal cortex, classically by an adenoma, results in increased sodium reabsorption by the distal renal tubule leading to extracellular fluid volume expansion and hypertension. This expansion is "sensed" by the juxtaglomerular apparatus cells and their production and secretion of renin are suppressed. The fall in renin and, subsequently, angiotensin II levels, however, does not result in decreased aldosterone secretion as it does in the normal, for, as noted, the aldosterone secretion is autonomous. It is the combination of the suppressed peripheral PRA and the fixed high aldosterone level that confirms the diagnosis of primary aldosteronism. Very rarely, an adrenal tumor can produce the precursor 11-deoxycorticosterone (DOC) instead of aldosterone and in these instances aldosterone levels will be low, but DOC levels will be elevated. Since aldosterone stimulated reabsorption of sodium from the distal tubule takes place mainly in exchange for potassium, hypokalemia becomes a prominent feature in most patients with primary aldosteronism and the measurement of serum potassium provides a simple screening test.

Other Documented Adrenal Cortical Diseases

Peripheral PRA also is suppressed in two other well defined situations where there is documented steroid hormone excess, sodium retention, and hypertension, namely Cushing's syndrome and certain forms of the adrenogenital syndrome. In Cushing's syndrome the salt retaining effect of large amounts of glucocorticoids offers an adequate explanation for the suppression of PRA, however, increased production of mineralocorticoids also has been found in some patients. In the C-11 hydroxylase deficiency form of the adrenogenital syndrome there is increased production of androgens and of DOC, and it is the DOC that causes sodium retention and PRA suppression. These patients present as masculinized females or precocious males with hypertension. In the C-17 hydroxlase deficiency form there is decreased production of both androgens and estrogens and, theoretically, increased production of both DOC and aldosterone. Actually, however, only the DOC levels have been found to be consistently elevated. These patients present with poorly developed secondary sexual characteristics and hypertension. Of course, it is obvious that the diagnosis of Cushing's syndrome and of the adrenogenital syndrome usually is made on other grounds than the suppressed PRA.

Suppressed Plasma Renin Activity in Essential Hypertension

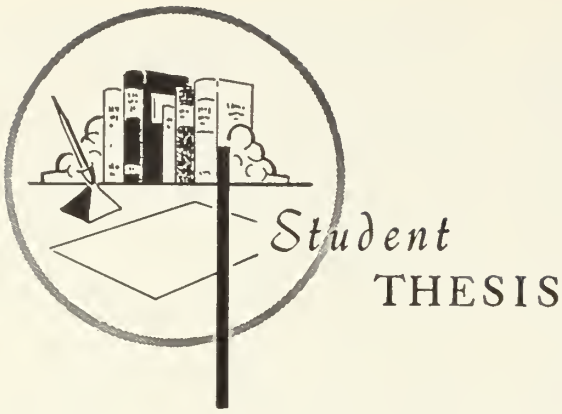
Jerome Conn postulated in 1964 that perhaps 20 per cent of patients who by all the usual criteria had essential hypertension actually had "normokalemic primary aldosteronism." This provided the impetus for investigators to measure peripheral PRA in large number of patients with hypertension. It now is obvious that at least 20 per cent do indeed have the suppressed PRA; however, as noted above, most of these do not have elevated or fixed aldosterone levels. Neither do any significant number have Cushing's syndrome or the adrenogenital syndrome. A great deal of effort is being expended at the present time in an attempt to determine why PRA is suppressed in the remainder. A defect in the sympathetic nervous system response to volume depletion has been offered as one explanation. It is more tempting, however, to implicate some unidentified type of mineralocorticoid excess in these patients. In support of the latter, it has been shown that they do have expanded extracellular fluid volumes and that the inhibition of adrenal steroid synthesis by aminoglutethimide or the administration of the mineralocorticoid antagonist spironolactone can result in sodium and water loss and lowering of blood pressure. A search for other possible mineralocorticoids has led to the discovery by Melby and his associates of increased secretion of 18-hydroxy-11-deoxycorticosterone in a few, but certainly not the majority, of these patients.

An important additional factor in attempting to understand the significance of a suppressed PRA is the contention by Laragh and his associates that the finding of a suppressed peripheral PRA has important prognostic implications apart from any possible relationship to etiology. Their data show that patients with hypertension and low peripheral PRA have a more benign course than those with normal or high peripheral PRA and that they are protected from the development of strokes and heart attacks. If this is confirmed by others it would not only offer another reason for the measurement of PRA in patients with hypertension, but also would imply that the treatment of hypertension should emphasize those antihypertensive drugs, such as alpha methyl dopa and propranolol, that suppress renin secretion.

Recommendation

The efforts to elucidate the significance of a suppressed PRA are still continuing and the answers are not all available. In the meantime, however, a practical guideline is needed as to when the clinician

(Continued on page 309)



Actinic Keratosis—The Advent of Fluorouracil

LARRY ANDERSON, M.D., *Kansas City, Missouri*

THE CHARACTERISTIC hyperkeratotic, flat, red papules on the exposed portions of the body are well known to dermatologists, internists, and family physicians. Although these lesions are often multiple and quite unsightly, their importance is not related to their cosmetic effect but to their strongly premalignant nature with tendency to develop malignant squamous cell changes. This paper is based on a review of the literature in regard to actinic keratosis (solar keratosis, senile keratosis). The comments on predispositions, pathogenesis, diagnosis, management, and treatment of actinic keratosis are oriented to information which the primary physician should know in order to render optimum care to patients with these premalignant lesions.

Incidence and Predispositions

The true incidence of actinic keratosis is unknown. This is also true of squamous cell carcinoma of the skin, because all skin malignancies tend to be recorded under the broad term of cutaneous neoplasms. It has long been recognized, however, that actinic keratosis occurs almost entirely in fair-skinned, blue-eyed, and often blond or red-haired individuals who have been exposed to sunlight for long hours. These people also have a strong tendency to freckle or burn rather than tan, when exposed to the sun's rays. Work by Fletcher in 1950 has shown that the predisposition to develop actinic keratosis seems to be more related to the eye color of the individual and his parents, than to hair color or apparent "fairness" of the skin. It seems that the stronger the inheritance for brown eyes, the less likely a person is to develop these skin changes. The blue-eyed individual with blue-eyed parents is more likely to develop actinic keratosis than is the brown-eyed individual with brown-eyed parents. The brown-eyed individual with one blue-eyed and one brown-eyed parent has intermediate susceptibility.

Dark-skinned races are very resistant to the solar-caused degenerative skin changes. The incidence of actinic keratosis induced squamous cell cancer in the Negro race is almost zero; however, the incidence of squamous cell lesions on non-exposed areas of the body are comparable in both the dark and light-skinned races.

Almost 60 per cent of the reported cases of actinic keratosis occur in patients between 50 and 70 years of age. In those individuals who are extremely susceptible to solar radiation, these skin changes will occur 10-20 years earlier. The Celtic-speaking peoples (England, Ireland, Scotland) are known for their marked predisposition to develop actinic keratosis. Some epidemiologists have proposed that due to the reduced amount of sunlight received in the British Isles, these people may not have developed the usual ability for skin changes (increased pigmentation and thickening of the stratum corneum) which provide some degree of protection from the sun's rays.

Pathogenesis

Actinic keratosis occurs due to a disturbed symbiotic balance in the relationship between malpighian cells and adnexal epithelium in the epidermis. These

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This is one of a group of theses written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the School.

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cells undergo heritable changes which make them functionally inadequate while developing tendencies for hyperproliferation, but which may lead through a progression to carcinoma-in-situ and occasionally to invasive carcinoma.

As early as 1894, Unna suggested that prolonged exposure to the rays of the sun might have an etiological relationship to the development of degenerative and malignant lesions of the skin. In 1906, Hyde questioned whether actinic rays of light might unfavorably influence certain sensitive skin in the direction of "epitheliomatous metamorphosis." Hyde had eliminated heat, wind, and frostbite as etiological factors due to geographical distribution. The idea of carcinogenesis due to light waves was supported in 1928, by the production of skin cancer in rodents following exposure to mercury arc lamps. Shortly thereafter, Findlay duplicated this experimental finding using chronic exposure to ultraviolet radiation.

The lower wave lengths of sunlight in the atmosphere are determined by a layer of ozone in the stratosphere. This layer starts to absorb wave lengths below 3200 Å (angstroms), with almost complete absorption of wavelengths below 2900 Å. It has been shown in both animal and human experimental models that the sunburn spectrum is in the ultraviolet range of 2900-3200 Å. This same spectrum has also been shown to contain the wave lengths active in carcinogenesis and in the antirachitic factors of Vitamin D.

It is well known that forms of energy can be interconverted, and for many years it has been postulated that the damage done in biological systems (skin) exposed to high energy radiation may be due to the formation of free radicals. A free radical is a chemical species with one or more unpaired electrons which make it a highly reactive molecule, by which ultraviolet radiation can exert a chemical change in the skin.

Two different free radicals have been discovered in epidermal tissue exposed to ultraviolet radiation of less than 3200 Å. Type I radicals seem to be generated by epidermal proteins and have been implicated in epidermal cell damage with dyskeratosis, epidermal cell disorganization, and rupture of lysosome and keratinosome membranes. Type II radicals are detected in the pigmented skin and appear to be generated by melanosomes of the epidermis. Type II radicals have been implicated in the oxidation of melanin and are probably active in the immediate tanning effect, which is seen during and shortly after exposure to ultraviolet light (immediate pigment darkening). For simplification, it may be considered that these free radical forms may cause the biological reactions necessary for skin tanning, the changes recognized as sunburn (erythema, blistering, and cell destruction), and those changes of actinic keratosis (dyskeratosis, acanthosis, and others), depending on the extent and chronicity of exposure to ultraviolet radiation. These free radicals have been found to produce biological changes by causing cell death, inhibiting cell division and cell growth, causing gene mutation, and by disturbing biological reactions such as the oxidation-reduction mechanism of melanin previously mentioned.

In vivo studies on human and mouse skin by Fukuyama, in 1967, have shown a marked decrease in cell mitosis and decreased synthesis of deoxyribonucleic acid, ribonucelic acid, and protein within 1-3 hours following ultraviolet radiation. Recovery from this decreased level of activity occurs within a few hours to more than a day postexposure, with a rather rapid acceleration of all these cellular functions to a peak of activity in 48-72 hours. There is then a gradual return to normal levels of activity in about seven days. Following chronic exposures to ultraviolet radiation, there is a continual stimulation of DNA synthesis and mitosis with a decrease in epidermal cell transit time. Cell loss falls behind cell production, resulting in acanthosis. These changes can be made to continue under experimental conditions until hyperplastic reactions give way to neoplastic changes and squamous cell carcinoma is produced.

Using guinea pig skin, it was found that one chromophore for absorption of ultraviolet radiation in mammalian skin appears to be epidermal DNA. The effect of this radiation on epidermal DNA seems to be related to the formation of pyrimidine dimers in the DNA molecule. In summarizing the pathogenesis of ultraviolet-induced changes in the epidermal tissues, "the most obvious responses of biologic tissue such as skin to light, may therefore be considered to involve photodimerization of thymine, uracil, and cytosine in the DNA and RNA of the epidermal cells, denaturation of enzymes, cell death, and mutation." These changes may be due to the direct effect of ultraviolet radiation or to the intermediary effect of the free radicals as discussed above.

Histology

Solar keratosis shows marked hyperkeratosis and parakeratosis with extension of the acanthotic epidermis into the corium, showing moderate papillomatosis. Epidermal cells show decreased basophilia, disorientation, and variation in size and shape. Some nuclei are enlarged and show prominent nucleoli and increased staining. Frequently, individual cells in the epidermis will be seen undergoing keratinization in the midst of the prickle cell layer where keratin

ordinarily is not seen (dyskeratosis). The lateral margin between the keratosis and adjacent normal skin is quite sharp, supporting the idea that "keratosis senilis consists of the progeny of one diseased cell that has acquired heritable changes leading it on the path toward malignancy." This line of demarcation is usually oblique, with keratosis extending under normal appearing epidermal cells on the surface of the skin. The basal cell layer is intact with a moderately dense lymphocytic infiltration in the upper dermis. There is a characteristic increase in dermal connective tissue which stains abnormally with elastic tissue stains (solar or actinic elastosis). Actinic elastosis is a very characteristic finding and probably causes the wrinkling seen on the surface surrounding lesions of actinic keratosis. The dyskeratotic and mitotic activity often gives a definite malignant appearance to solar keratotic lesions, and for this reason some pathologists consider this a grade one-half malignancy.

Diagnosis

The diagnosis is usually of no particular problem because of the characteristic superficial, red, scaling keratosis found on the exposed skin of middle-aged, fair-skinned patients with a history of excessive exposure to ultraviolet radiation. These lesions are usually multiple and vary in size from microscopic to 1 cm in diameter. Lesions larger than 1 cm are rare without invasive squamous cell carcinoma. The surrounding skin is usually yellow to brown to black and furrowed due to the underlying actinic elastosis. Biopsy should be performed in any questionable case, in most lesions with a cutaneous horn due to the high occurrence of malignant changes in the base of the horn, and in any lesion which has suddenly become larger, more painful, or started to bleed without trauma.

Following the example of the gynecologists who have used vital staining of cervical lesions, recent use of 1 per cent toluidine blue dye has been used as a vital stain by some dermatologists dealing with suspected skin neoplasms. One review of this technique recorded positive results in 17 of 18 malignant and 6 of 7 premalignant squamous cell lesions proven by histology. Vital staining is not expected to replace biopsy as a means of diagnosis, but it can be of value in delineation of the complete lesion, in identifying probable positive lesions when many are present, and in the early diagnosis of recurrence in a previously treated site.

Prognosis and Treatment

Actinic keratosis is definitely a premalignant lesion with 15 to 25 per cent of the patients developing one or more squamous cell malignancies if left untreated. However, it should be mentioned that these malignancies are not as clinically malignant as are those squamous cell lesions arising de novo. For example, only 0.1 per cent of the malignant lesions arising from actinic keratosis will show signs of metastasis. Due to the early visibility, slow growth, easy diagnosis, and good forms of treatment these lesions should be 100 per cent cured.

There are four primary types of treatment for actinic keratosis:

1. Surgery
2. Curettage followed by destruction of the base
3. X-ray or radium therapy
4. Chemotherapy (5-Fluorouracil)

The first three forms of therapy have been used for many years, are well known, and will receive no further mention. Chemical peeling and dermabrasion are two other treatments which have been used by some physicians with no clear-cut advantages. Several chemotherapeutic agents have been used in the treatment of actinic keratosis, but one has been so superior that it has really eliminiated all others for general use. Following its synthesis in 1957, the observation was made that solar keratosis cleared in patients who were receiving 5-Fluorouracil for internal malignancies. Due to significant systemic toxicity from parenterally administered 5-FU, topical administration was attempted and the results have been so good that most physicians now feel topical 5-FU is probably the best treatment available for actinic keratosis, especially with multiple lesions.

Strictly controlled studies with 5-FU have shown that this chemotherapeutic agent, when applied topically, produces no changes in normal skin, but selectively attacks actinic keratosis. There have been no reported systemic side effects from the topical use of this drug and the only untoward effects have been an occasional conjunctivitis due to local irritation, one reported case of bullous pemphigoid in an 84-year-old patient, and a 1+ per cent incidence of photosensitivity. The case of bullous pemphigoid showed typical skin lesions and positive basement membrane antibodies in serum and blister fluid. This patient responded well to hospitalization and steroid therapy over several months with a disappearance of the skin lesions and antibodies. One half of the reported photosensitivities have shown on patch test to be due to the vehicle used, with the other half apparently due to 5-FU.

The mechanism of action of 5-FU has been shown to be the in vivo inhibition of thymidylate synthetase. This inhibitory action was measured in actinic keratosis and in normal skin, with the findings of partial to complete inhibition in all actinic skin tested compared to only partial inhibition in 50 per cent of the normal skin. The efficacy of topical 5-FU can

be increased by the use of occlusive dressings and by the exposure to ultraviolet radiation either during or immediately prior to initiation of therapy.

The 5-FU is routinely prepared as either a 1 per cent or 2 per cent solution in propylene glycol, or as a 5 per cent concentration in a hydrophilic petrolatum base. The medication is then applied to the affected area twice daily with a two-week treatment program for the face, neck and trunk, or a four-week program on the forearms, hands, legs, and feet. The patient should be seen at least weekly by the prescribing physician, and the rate of application may be altered if the skin reaction seems excessive. The erythematous reaction of the skin occasionally causes moderate discomfort and steroid ointments may be used, along with several days' rest from treatment if necessary. Steroid ointments are often used after the treatment program and the scaling and erythema rapidly subside in two to three weeks with remarkably good cosmetic results.

The specific activity of 5-FU against actinic damaged skin may be due to the local disruption of the epidermal barrier, providing an avenue for increased absorption of 5-FU. Once through the barrier, 5-FU acts to inhibit nucleic acid synthesis leading to interference with cellular functions and an altered mitotic rate. This results in an inflammatory reaction which develops in proportion to the degree of preexisting sun damage and usually reaches a peak in 7-10 days. Histological examination of the treated skin reveals swollen acantholytic epidermal cells with distorted nuclei. An intense mononuclear inflammatory infiltrate develops with subsequent sloughing of the reaction site, leaving a relatively unaffected appearing dermis which will heal without scarring in about four weeks. The patient may well have a recurrence of actinic keratosis at some time in the future, but he can expect two to four years of freedom from this problem and can usually be treated again with 5-FU if necessary.

Easy medicine to apply, with good patient acceptance, 5-FU is inexpensive and safe. This agent is especially good on extensively involved areas because it will search out and destroy not only the large lesions, but also those lesions which are too small to recognize. It should be kept in mind that large indurated lesions and lesions containing squamous cell carcinoma will respond in only about 50 per cent of the cases, and these lesions will then require some further mode of therapy.

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Clinical Cardiology

(Continued from page 305)

should attempt to demonstrate, with sodium restriction and ambulation, suppressed peripheral PRA. In formulating this guideline, the important point to emphasize is that at the present time the measurement of peripheral PRA, in a patient who by all other criteria has essential hypertension, probably will lead neither to a cure of the hypertension nor to a drastic change in therapy. This being the case, it is recommended that for the present the attempt to demonstrate suppressed PRA be limited to those patients who have features that suggest the presence of the specific entity, primary aldosteronism. The most important feature, of course, is spontaneous hypokalemia. As implied above, this recommendation may change drastically if further causes of suppressed PRA are defined or if the prognostic significance is confirmed.

NEW MEMBERS

The JOURNAL *takes this opportunity to welcome these new members into the Kansas Medical Society.*

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Socio-
ECONOMICS

Williams-Steiger Act

Occupational Safety and Health in Kansas

LEE ELLIS, M.S.* *and* IRVIN G. FRANZEN, M.P.H.,† *Topeka*

In December 1970, the President signed into law the Williams-Steiger Occupational Safety and Health Act of 1970. It became effective in April 1971, and its fundamental objective is contained in the provision that "each employer has the basic duty to furnish his employees employment and a place of employment which are free from recognized hazards that are causing or are likely to cause death or serious physical harm."[1] This legislation provides the framework for the most extensive effort ever undertaken to reduce the over 14,000 fatal occupational injuries, and the estimated two million disabling injuries[2, 3] and 150,000 disabling occupational diseases[4, 5] that have been occurring among 80 million American workers annually. Among the provisions of the Act are those allowing each state to assume most of the administrative functions. Some of these functions have already come under Kansas control; others, if they are to be assumed by the state, will require special legislative action that will not occur in the immediate future.

This paper describes the basic features of the Williams-Steiger Act, where it seems to fit within a public health approach to injury and illness prevention, and how it may help to augment the efforts of physicians and other health personnel working to improve the health of Kansans. Since many physicians in private practice employ nurses or other assistants and may not be wholly aware of the Act's employer requirements, a purpose of this paper also is to outline the aspects of the Act that particularly relate to physician's offices and hospitals.

Background

The passage of the Williams-Steiger Act comes in the wake of three trends in the field of health. The first is the growing pressure for greater coordination in nearly all areas of health maintenance and health care delivery.[6, 8] Many efforts are under way to reassess America's health system so that the available manpower may be utilized more effectively. The problem does not appear to be simply a shortage of physicians; some are suggesting that the entire health system needs to be reevaluated.[9] This seems particularly warranted in light of the possibility that there may not be a strong relationship between the health of community residents and the ratio of physicians per capita.[10]

The second trend is that of an increasing emphasis upon preventive approaches to health.[11] With the demands upon the physicians' time becoming increasingly acute, preventive approaches are appearing as an attractive adjunct to direct medical treatment. Preventive approaches have been found to provide an opportunity for many persons to con-

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tribute in a significant way to the health field with considerably less formal training than that required for physicians; and, in the long run, preventive health care should save not only human suffering but also public expense.[12]

The third significant public health trend is that accidental injuries increasingly are being viewed from an epidemiological perspective. This perspective postulates that accidental injuries, like most illnesses, can be significantly reduced through coming to understand them within the "susceptible host, predisposing environment and inciting agent" interaction model.[13] Successful intervention within the scope of any one of these interrelated factors should have an effect upon the resulting injury or illness rate for a given population.

Overview

The main emphasis of the Williams-Steiger Act is upon the prevention of occupational injuries and illnesses. However, the act also contains certain provisions to more adequately insure that employees who sustain an injury or illness will receive useful first aid and have quick access to professional medical treatment when needed. Many businesses, especially those in construction trades where medical facilities are sometimes remote, will be required to provide a number of their employees with basic first-aid training. Employers may come to a physician for advice in this regard; if they do, a physician may recommend that the employer contact the Midwest Center of the American Red Cross, 4050 Lindell Boulevard, St. Louis, Missouri 63108. A new, highly structured, one-day course in first aid has been developed by the American Telephone and Telegraph Corporation in cooperation with the American Red Cross,[14] which has been widely commended for the amount of useful emergency treatment information it provides. Also, in accordance with other requirements of the Williams-Steiger Act, a number of employers will be contacting one of their community physicians to arrange a standing agreement regarding the unencumbered processing of all medical emergencies involving their employees.

These types of requirements are to help reduce much of the confusion over how to proceed in case of sudden emergency, and are in line with studies which have found that quite a number of permanent disabilities and deaths from physical injuries could be avoided if prompt and systematic emergency procedures are followed when accidents occur.[15-17]

The Williams-Steiger Act is being administered at the federal level principally by two agencies. One is the Occupational Safety and Health Administration (OSHA), under the US Department of Labor, whose main function is that of coordinating all inspection efforts. The other is the National Institute of Occupational Safety and Health (NIOSH), under the US Department of Health, Education and Welfare, whose duties are primarily those of conducting the basic laboratory research needed for improving the safety and health standards.

The prevention of occupational injuries and illnesses under the Act is being approached in three ways. The first of these involves the monitoring of injury and illness rates through an annual survey of business and industry. Nationally, approximately one-fourth of one million businesses will be surveyed each year; in Kansas, about 8,000 businesses will be surveyed. Each year's sample will be chosen at random within each business category, with varying probabilities depending upon employment size. The necessity of these recordkeeping and surveying provisions may not be apparent to physicians in private practice who are in the enviable position of being able to assess the effectiveness of their efforts on the basis of the one-to-one relationship they establish with each of their patients. Those who work to improve health from a preventive or public health approach cannot use such a criterion; first, because they will never have contact with most of those they are trying to help, and second, because they could not directly "see" effective prevention even if it were possible to meet with every individual concerned. Therefore, the only way it will ever be possible to determine to what degree the basic objectives of the Williams-Steiger Act are being achieved is through periodic sampling of data from records that every business establishment keeps.

As a second pursuance, OSHA will conduct actual inspections to attempt to identify hazards which violate safety and health standards. States may submit a plan for assuming the inspection authority of OSHA. Whether or not Kansas eventually will assume this authority is not known. In the latter part of 1972, a legislative study committee advised against a proposal for assuming the inspection and enforcement authority of OSHA. Certain revisions of the proposal were considered, but at the year's end hints appeared that some substantive changes in the Act may, at the federal level, be forthcoming. Subsequently, all additional proposals regarding state control of the inspection and enforcement authority of OSHA have been formally withdrawn from consideration in the immediate future. If such a plan is approved, the states may modify the standards in accordance with their perceived needs, as long as minimum federal conditions are maintained. Otherwise, the inspection and enforcement authority will continue to rest with federal officials operating out of Kansas City, Missouri.

The third approach will be through the conduct

of basic research on general product and equipment safety, and other studies of the physiological and behavioral factors relevant to safety and health in an occupational setting. Information gained in this way will be used to suggest improvements in standards used by the inspectors. Most of the more basic research will be confined to NIOSH, and private organizations to whom research contracts are awarded. However, state agencies who are delegated to conduct the annual occupational safety and health surveys (in Kansas, the State Department of Health), if their schedules permit, may also contribute to the more basic research aims through various special studies. In Kansas, we are undertaking a special study of public and private businesses to determine if occupational injuries are partly the function of when and how often worktime is lost due to sickness or vacations. Previous studies indicate that automobile accidents are somewhat more prevalent among those suffering with various illnesses, including diabetes, epilepsy, cardiovascular illnesses, and mental disorders;[18, 19] and that work accidents are greater among alcoholics.[20, 21] Another report presented evidence that injuries in general are more probable for workers who manifest virtually any symptom of ill health.[22] Although scientific evidence appears to be lacking, at least one writer has asserted that taking a vacation may help to lower one's sickness probability in general (Rokstir, *Today's Health,* 99:442, 1957).

Inadequacies of Previous Legislation

Spokesmen, both in Kansas[23] and in the nation,[3, 24] have generally considered previous occupational safety and health legislation inadequate for many years. The problems have been complex and are difficult to summarize; this critique will pertain only to the more fundamental difficulties, and describe in general terms how the Williams-Steiger Act has been designed to deal with them.

In Kansas, as in a number of other states, the enforcement of occupational safety standards has been handled under the State Department of Labor, while occupational health has been legislatively delegated to the State Department of Health. Inspection duplications have been inevitable, and inadequate statutory authority has made it difficult for state officials to require negligent companies to improve their safety and health condition once hazards were identified. If Kansas does assume the inspection and enforcement functions of OSHA, the Williams-Steiger Act will require both of these state agencies to reassess and coordinate their safety and health efforts, both in regard to inspection and enforcement of standards, as well as in regard to their use of statistics in evaluating the effects. No statistical evaluations of the state's enforcement efforts had been undertaken before the Williams-Steiger Act requirement that employers keep accident records.

It has been well established that the correction of any public health problem is virtually impossible without reliable knowledge of the degree to which the problem exists within various segments of the population and the conditions associated with it.[25-27] Ideally, one will continue to obtain this knowledge periodically, so that the effects of any remedial attempts will be apparent. With an average of only around 95 occupational deaths annually taking place in Kansas in recent years, generalizations about any year-to-year trends by type of industry are virtually impossible because of random fluctuations in such small numbers. Therefore, more information than mortality experience is needed to assess the effectiveness of the efforts of safety and health inspectors. The Workmen's Compensation Office of Kansas receives around 18,000 injury reports annually. Fewer than 30 per cent of Kansas workers, however, come under compulsory coverage, and the Workmen's Compensation Office has not had the personnel or facilities to code and analyze the reports they receive much beyond what has been required to insure fair payment to injured workers. The Williams-Steiger Act now requires that essentially all employers maintain occupational injury and illness records and that, upon request, these records be made available in a uniform way to OSHA representatives.

Some states have maintained annual statistics on occupational injuries beyond those resulting in death, to help monitor their safety inspection efforts. These have been combined each year, by the US Bureau of Labor Statistics, with other figures BLS has independently collected from some additional cooperating industries nationwide. However, since all of these occupational injury records were kept on an essentially voluntary basis,[3] obvious sampling biases have made reliable generalizations very difficult. For example, the reporting companies tended to be much larger in terms of the number employed than those who did not report their injuries;[28] and a number of studies have found larger business establishments on the average to have considerably lower accidental injury rates than smaller establishments performing comparable functions.[28-32] The one exception to this appears to be in the field of farming.[33]

Another flaw in the old record keeping system, as it was used to obtain national figures, had to do with its lack of provisions for recording injuries followed by a job transfer. Under the old system, if an employee returned to work the day after an injury, no matter how menial his changed job assignment may have been while he recovered, his injury was not recorded; under the OSHA system it will be.

Trends

Throughout the 20th century, a considerable amount of scientific research has been directed toward the prevention of work injuries.[34] Although the data and study designs have not been as methodologically pure as those found in most tightly controlled medical experiments, industrial safety and health studies have reached a considerable number of generalizations for preventing occupational accidents. One would expect, therefore, that some improvement in the safety and health for the American work environment should have taken place over the years. The only reliable annual statistics compiled in Kansas and uniformly across the nation are on fatal occupational injuries. All other figures are merely estimates derived from special studies in which a certain number of nonfatal injuries or occupational diseases have been found to occur for every fatal injury. For example, some limited studies under the old reporting system revealed that there was an average ratio of 155 nonfatal injuries for every fatality.

Even though there is little uncertainty about the total number of occupational fatalities each year, such figures are difficult to interpret with regard to the relative safety of today's work environment compared to that of past years. *Figure 1* shows that the rate of occupational accidental deaths per 100,000 employed persons nationally has been reduced by half, and that the Kansas rate has been reduced by three-fifths over the past 30 years. Fatal injury rates, however, computed simply in terms of size of the work force fail to allow for the following factors.

First, the average hours worked per employee have decreased about 18 per cent during the period of 1940-1970.[35] Second, the average number of paid vacation days and holidays allowed American workers has greatly increased over the same period, perhaps as much as 50 per cent.[36] These two factors directly reduce the likelihood of occupational injuries by lowering the total number of exposure hours and, presumably, may have had some indirect influence by lowering workers' tendency toward greater carelessness due to fatigue or boredom.

Third, a somewhat greater proportion of workers now are in white collar, lower-risk occupations than were 30 years ago.[37] Finally, fewer extremely old and extremely young employees are now in the labor force.[38] Young employees especially have been found to have a greater likelihood of accidental injuries.[39]

Although no precise adjustments for these qualifying factors are possible, it is our estimate that they account for no more than 70 per cent of the gradual decrease in fatal occupational injuries which have taken place since 1940. The remainder of the decrease is presumably explainable in terms of improved physical work conditions or safer employee work practices, which the research in industrial safety and health has helped to promote.

Compliance Requirements

The OSHA safety and health standards have been the source of the greatest concern and uncertainty with regard to the Williams-Steiger Act. This section will, therefore, present an overview of these require-

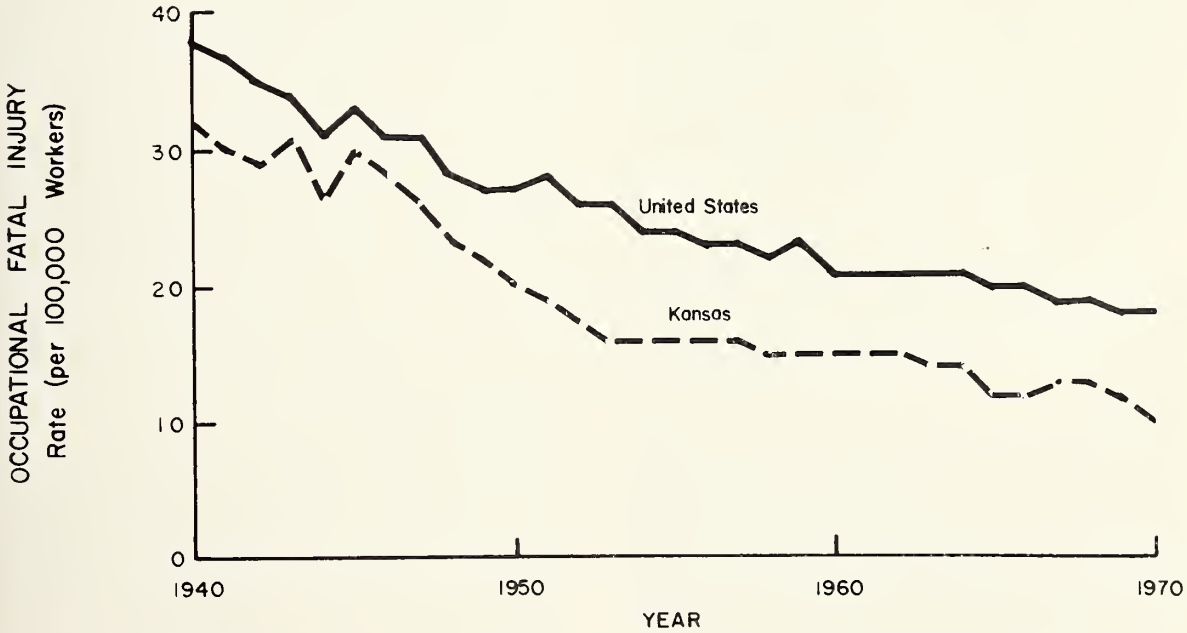

Figure 1. The annual rate of fatal occupational injuries per 100,000 workers (United States and Kansas, 1940-70).

ments from the standpoint of how they are actually being enforced. The occupational safety and health standards were written to reflect the recommendations of private industrial organizations and various non-profit safety and health associations, such as the National Safety Council, the National Fire Prevention Association, the American National Standards Institute, and the American Public Health Association. Already, a number of these standards have been found impractical or in need of modification, and have subsequently been deleted or qualified. Reports by many appointed study committees, consisting of government and private industry representatives, are being considered. Several check-and-balance provisions are outlined in the Act to protect employers against unfair charges of compliance violations, excessive imposition of penalties, and unrealistic abatement periods.

In light of the fact that safety and health are inherently relative,[40] it is unrealistic to expect that the standards will ever stabilize. In underscoring the relative nature of the very concepts of safety and health, Michels recently stated, "What is safe today is predictably unsafe tomorrow, even if the safety standards were to remain constant."[41]

No physician's office and no hospital in Kansas has yet been inspected by OSHA representatives. Business operations in the health service field are traditionally among the least hazardous from the standpoint of the employees,[2, 42] and their probability of being inspected by OSHA agents in the future is correspondingly low. The types of potential hazards common to both that would come under the most careful scrutiny would include those pertaining to fire safety, radiation and chemical storage safety, and appropriate precautions against unauthorized access to medications. For additional ideas on the types of hazards to which health service personnel may be exposed, Mammen has written an excellent article based on extensive observations in a large general hospital.[43]

Anyone in doubt about the specific applicability of any safety and health requirements to their business location may contact the OSHA Regional Office, US Department of Labor, 911 Walnut Street, Kansas City, Missouri 64106 (Phone: 816-374-5249). An inquiry to this office is legally required to have no effect upon the probability of being inspected. Once an OSHA inspector has arrived at a business establishment, however, violations are subject to penalty at that point. Of the 270 inspections conducted in Kansas in the 1972 fiscal year, 240 businesses were found to have had at least one standards violation. The proposed penalties averaged $71 per business, or an average of $26 for each violation cited.

Under unusual circumstances, an employer may wish to contract the services of a private safety consulting firm on a fee basis. One such firm already operates in Kansas. It is, however, strongly recommended that any such contractual arrangement by an employer be preceded by a call to the OSHA Regional Office for some idea of the nature and extent of the safety and health compliance problems that may exist.

Eventually, OSHA will publish separate compliance manuals for each major business category. Presently, the only category for which a separate manual is available is construction. All other businesses are subject to the guidelines of the "General Occupational Safety and Health Standards" manual, published in the *Federal Register* 35(105):10466, 1971.

Recordkeeping Requirements

Table I and the following discussion summarize the employer's recordkeeping responsibilities under the Act *if* he has eight or more employees. First, complete instructions and forms are contained in a booklet entitled "Recordkeeping Requirements under the Williams-Steiger Occupational Safety and Health Act," which is available from the OSHA Regional Office, or the Occupational Safety and Health Survey Office, Kansas State Department of Health, Topeka, Kansas 66603 (Phone: 913-296-3523). The booklet's centerfold consists of a poster which every business establishment must display for its employees, informing them of the purpose of the Occupational Safety and Health Act and their rights to file complaints of unhealthful working conditions.

Second, all recordable injuries and illnesses are to be entered upon OSHA Form-100 (the basic log), and then are to be more fully described on OSHA Form-101. In Kansas, employers may substitute for this the Kansas Workmen's Compensation form.

Third, at the end of every calendar year, the employer is required to complete the OSHA Form-102, an abbreviated summary of the company's injury and illness record. If no occupational injuries and illnesses occurred, all that needs to be done for the entire year is to indicate "none" across the Form-102.

The Form-102 is to be posted for employee inspection on or before the end of January each year, and remain posted for not less than 30 days. The Form-102 is the only one to which employees are required to have access.

Finally, on a sampling basis, every February, this office will mail a final form—called the OSHA Form-103—to a sample of approximately 8,000 businesses. On the back, the format of the Form-103 is identical in large part to that of the Form-102; on the front there are approximately ten questions concerning the nature of the business, average employment, total employee hours worked, and the types of basic medi-

TABLE I
OUTLINE OF OSHA RECORDKEEPING REQUIREMENTS

Forms	*Description*	*Action Required*
Centerfold poster	Notification to employee of the Act's requirements	Post where employees usually read notices
OSHA Form-100	Basic log of injury or illness	Line entry for each injury or illness; file
OSHA Form-101 (Kansas Workmen's Compensation Forms may be substituted)	Detailed supplement to the log	One-page entry for each injury or illness; file
OSHA Form-102	Annual summary	One-page entry for all injuries and illnesses for each calendar year; post for thirty days for employee inspection each January; file
OSHA Form-103 (Only to be completed if notified by mail to do so)	Survey form	One-page entry for all yearly injuries and illnesses plus one page of "control information"; return form to survey office; file retainer copy

cal facilities and personnel available to employees. Approximately 100 physicians' offices, clinics, and hospitals will receive Form-103 this year.

A recent amendment to the Williams-Steiger Act (*Federal Register,* 37(193):20823, 1972) exempts all businesses with fewer than eight employees in a given year (beginning with 1973) from all recordkeeping requirements. This is with the exception of a basically random sample, who will be notified otherwise by mail at the beginning of a particular calendar year. Upon such notice, these small businesses will only need to complete two forms: the basic log (OSHA Form-100), and the survey form (OSHA Form-103).

One question that came to the survey office last year had to do with whether or not physicians are to include themselves in the employment figures. The answer is generally no. The only conditions under which a physician would be included are if he received a regular salary and was not instrumental in deciding his work schedule and duties, such as at a hospital. In these cases, the hospital would keep the OSHA records, not the physician. In those cases where an arbitrary decision is required, the important point is to make sure the same employees are covered in the work time estimates as in the injury and illness records. To avoid bias, all "employees" (versus "employers") should be so designated before, rather than after, an accident befalls them.

Since the less severe accidents are most prevalent and injury severity is quite often a matter of "luck," the recording of minor (nondisability) injuries in addition to the more serious ones provides the best information for discovering effective preventive measures.[27] For this reason, OSHA requires the recording of three basic categories of injuries and illnesses: fatal cases, cases resulting in lost workdays, and nonfatal cases resulting in no lost workdays. Accidents in which a workman loses only what time remains in the day of injury itself should be considered non-lost workday cases. Otherwise, non-lost workday cases should include those resulting in the loss of consciousness, the restriction of work or motion, or medical treatment (except treatment of a purely precautionary or preventive nature). The only occupational injuries or illnesses not to be recorded are first aid cases. First aid has been defined as simple one-time treatment and subsequent observation of minor scratches, cuts, or burns for which medical care is not normally required.[44] The distinction between first aid and medical treatment should not be made on the basis of who administers the care, but on the basis of the nature of the care itself.

Conclusion

Among the factors vital to the success of the Williams-Steiger Act is employer awareness of its basic requirements. In Kansas, we are attempting to provide information to employers in every way possible, and have been encouraged by the atmosphere of cooperation generally accompanying the responses we have received. In turn, we are trying to convey

(Continued on page 326)

The President's Message

I saw this article in the paper and was so impressed, I wanted to share it with you.

> My office will be closed until the middle of July and the appointments are filled. Will be in the office during August.
>
> Medical practice is no longer like it used to be. Federal regulations are a pain in the neck. In Payment Plans, they say you don't need care from one to three months. If you need it while I'm gone, write your Senator or Congressman about the plight of rural practice. If he fails to answer within thirty days, check with the local hospital to see if they are still allowed to take care of your emergencies.
>
> YOU are now a political pawn, so take care of yourself as you learned to do in "Scouting days." Uncle Sam doesn't really care about your health as long as you are strong enough to send in your taxes.
>
> Good Health,
> Charles R. Svoboda, M.D.

Thomas F. Taylor

President

Sibling Rivalry

The care and feeding of a medical journal generally receives little attention from any except the editorial board of that journal. The problems of attracting enough income to keep the journal viable has greater interest for its custodians than for the reader. Some ponderings along this line prompted the remarks we made a few months ago about the part the pharmaceutical manufacturers play in supporting medical education as accomplished by such periodicals. Realizing that the pursuit of the matter may be less than enchanting to some, we nonetheless press on in the hope of gaining some perspective on the problem. The only justification for taking this out of a board meeting and presenting it to the readers is that ultimately it is the readership (both qualitative and quantitative) which can be translated into a mechanism for the attraction of advertisers.

Of concern at the moment are the relative places of the subscription journals (in particular the regional journals such as this one) and the mass circulation journals, known irreverently and not always accurately as the throwaways. As noted before, the combination represents a formidable array of medical literature, which is good inasmuch as it permits the medical reader to select his choices from a remarkable smorgasbord of medical fare. The problem, however, lies in the fact that, having ingested a given selection, he may feel he has not made the best selection but has neither the time nor the capacity to go back for a helping from another plate. This, it seems to us, is the area of contention between the subscription journals and the throwaways: the physician's time and attention. In translation, this means that we are competing for the advertiser's dollar since this pays for the spread, and the pharmaceutical manufacturers intend that we take proper note of the table decorations they have provided.

A few months ago, one of our editor-colleagues sounded a call to arms to do battle against the throwaways because of the disproportionate financial blessings (reportedly 4 to 1) bestowed on them by these advertisers. We never got around to getting our uniform out of the cosmoline, but if this should come to his attention, he will know that despite the lack of formal response on our part he precipitated a little more than the usual thought on the matter.

The battle, in our interpretation, should not be a battle at all because of the differing natures of the two publications. This is not to say that we would not welcome a larger slice of the advertiser's pie. In fact, we hope the concern expressed by devoting this time, thought, and ink to the matter will lead to some means of accomplishing just that.

It comes down to the fact that the pharmaceutical companies want to sell their pharmaceuticals, which is understandable. The rules of the market place demand that they apply their efforts in the direction which will bring them the best return for their investment. It behooves the subscription journals, therefore, to do the same by pressing whatever peculiar advantages they may offer in the promotion of the success of these efforts.

It would be easy to mount an attack on the mass journals if they were of consistently poor quality or deficient service. The fact is that many are quite good, some even excellent, and they obviously serve a purpose other than simply being vehicles for pharmaceutical advertising. But this purpose is not the same as that of the regional subscription journals. For the most part, they provide abstracts, symposia, and reviews. They tell you what to do with your money (which you may or may not have) or leisure time (which you may or may not have). Some supply original articles and editorial comment of high value. Some, of course, contribute little of worth, and the great number of them results in some inevitable repetition, but subscription journals are vulnerable to the same charge. We can see no virtue in an effort

to eliminate any one or the group of them. This should be determined by the quality of their offering and the success of their function.

But what about the fact that the money spent on the support of throwaways is money not being spent on the subscription journals? The implication is that the removal of the competition would bring more money into the regional journal coffers and, further, that this would increase their quality. This is, at best, conjectural. Our thought is that the two are differing types of periodicals providing different services and the only area of competition is the attention of the reader.

The regional journal has certain inherent limitations of volume and appeal. For example, the 2,600 circulation of this journal is not going to increase appreciably unless we have a dramatic influx of physicians into Kansas, which doesn't seem likely. The other mechanism by which our readership might be increased would be a qualitative improvement of such a degree that there would be a nationwide demand for subscriptions and, even with a total replacement of the Editorial Board, this, too, seems unlikely. We have no illusions about the fact that we carry some items (only *some,* of course) that are less than deathless medical classics, but by and large we are providing a form of communication for the physicians of Kansas which, whatever its shortcomings, is their own. A few years ago, another editor-colleague proposed combining several of the neighboring state journals into one with a view to upgrading the content and improving the financial situation. We rejected the idea because, even if it were successful in these areas, it would eliminate whatever element of local appeal and character we now enjoy.

What about the financial realities? The advertising page rate of the more popular throwaways is as much as 30 times what ours is. But their mass circulation results in a cost per thousand to the advertiser of about half what ours is. It is not difficult to see which is more attractive to the advertising manager who wants to get his message across to the greatest number at the lowest cost. One might wonder why they bother with us at all, but it comes down to the fact that some consider it of value that the physician will see the message in his state journal and appreciate that it is a contribution to the existence of that enterprise.

If a significant increase in subscription volume seems impossible and a significant change in scientific and editorial appeal is at least problematic, what courses are open to such a journal? The subscription rate can be increased up to the point that the subscribers rebel and cancel. The advertising rate can be increased up to the point where the advertisers rebel and withdraw. Or it can improve its value to the advertiser by promoting increased use of the products by the readership until the public rebels at the number and cost of the pills it is taking (and we may be seeing that now). Or it can promote the effort of its readers to express to the advertisers their recognition of the contribution the advertising is making to the support of the publication. This points up the one significant difference between the mass and subscription journals. Whether good or bad, the former do not have the relationship to the individual reader that his regional journal has.

The mass journals aren't going to go away and the very fact of their large controlled volume is going to continue to account for their receiving the larger portion of the advertiser's money. But mean-

(Continued on page 322)

115th Annual Session

KANSAS MEDICAL SOCIETY

May 5-8, 1974, Topeka

MODERN ASPECTS OF TRAUMA

1. **Aeromedical Transportation of the Injured**
2. **Medico-Legal Aspects of Trauma**
3. **Symposium on Fat Embolism**

The 1974 meeting will be held in conjunction with the following specialty societies:

KANSAS SOCIETY OF PATHOLOGISTS
KANSAS OBSTETRICAL AND GYNECOLOGICAL SOCIETY
KANSAS ALLERGY SOCIETY
KANSAS SECTION ON EAR, NOSE AND THROAT

KMS Obituaries

CHARLES O. HOOVER, M.D.

Dr. Charles O. Hoover, 93, of Quinter, died May 30, 1973. He was born July 15, 1881, near Lone Star.

Dr. Hoover was graduated from the Eclectic Medical College, Kansas City, in 1910, and practiced medicine in Kansas for 55 years.

Surviving Dr. Hoover are his wife, two daughters, and three sons.

GEORGE A. PATTON, M.D.

Dr. George A. Patton, 85, of Atchison, died April 15, 1973. He was born in Topeka, in 1888.

Dr. Patton was graduated from the Meharry Medical College, Nashville, in 1918.

Surviving Dr. Patton is a daughter.

J. ALBERT WHEELER, M.D.

Dr. J. Albert Wheeler, 74, of Marion, died May 13, 1973. He was born in Marion, February 5, 1899.

Dr. Wheeler was graduated from the Eclectic Medical College, Cincinnati, in 1926. He had practiced medicine in Kansas since that time.

Surviving Dr. Wheeler are his wife, a daughter, and a son. Memorial contributions may be sent to the Axtell Christian Hospital, Newton, or the St. Luke Hospital, Marion.

AMA Committee on Community Health Care

Statement on Free Clinics—1972

FREE CLINICS, a form of health care delivery, have evolved in the last five years. They are new in the sense that they differ from the traditional established patterns of health care delivery practices.

The word "free" as used to describe this form of health care delivery is not limited to economics since many free clinics require some payment for service, but primarily denotes flexibility that allows rapid adaptation in the organizational structure, method of operation, and scope of services to meet individual patient and community needs.

The free clinic is *not* easily defined; however, they are characterized by several common and fundamental criteria:

1. Services are provided directly to all who seek them regardless of their ability to pay. In most cases there are no set charges, but donations are often requested.
2. In addition to professional health services, community and social assistance services are usually provided on an individual or a group basis.
3. The organization lacks conventional structure—there are not the usual regulations, eligibility requirements, administrative procedures, detailed record keeping, etc.
4. Health professionals frequently volunteer to serve without regular compensation.
5. There is little hierarchical difference among health workers.

There are generally three types of free clinics: "hippie," "neighborhood," and "youth"; however, a free clinic may be a combination or consolidation of these forms. The Haight-Ashbury Free Clinic, reportedly the first free clinic in modern times, was established in 1967 in San Francisco to provide drug treatment services to the drug-oriented youth subculture. This was the forerunner of those free clinics set up to serve patient populations having a high incidence of socially stigmatized diseases, such as drug addiction and venereal disease. Subsequently, free clinics have been established for the poor, the socially disenfranchised, and those who feel they cannot obtain adequate medical care through the more usual channels. For this segment of our population who might not otherwise receive *any* health care, the free clinics are filling a real need. These clinics provide a wide variety of health and health-related services not limited to drug dependence and V.D. treatment. Over 200 clinics in more than 30 states have been established.

Some free clinics are well staffed with physicians, nurses, and other health personnel and are adequately equipped to provide high quality health care. Others, however, have experienced staffing and operational difficulties and admittedly are providing limited services of less than optimal quality. Many clinics recognize that they have deficiencies of varying degrees and generally are attempting within their limited resources to make improvements. Examples of deficiencies are lack of continuity in care, inadequate records, and no referral mechanism for specialty and hospital services.

The free clinic movement appears to be gaining momentum. During the past 12 months it has been estimated that over two million patient visits have been made at free clinics. The AMA receives a number of inquiries concerning its position on free clinics. This subject has been examined at length by both the Committee on Community Health Care and the Committee on Health Care of the Poor. Because of the medical profession's recognized responsibilities for improving medical care for all persons, the Council on Medical Service offers the following recommendations:

1. In recognition of the fact that there are an increasing number of free clinics providing varying degrees of medical and health care in settings acceptable to only some population segments, organized medicine at the national, state, and local levels should continue to provide assistance and work to improve the quality of care in such clinics.
2. Local medical societies could improve liaison by having members or staff available to offer advice and assistance in the development or improvement of medical and health services in free clinics.
3. To assure quality medical care to all segments of our society, it is hoped that individual physicians and other health professionals will cooperate with those free clinics that are striving to give adequate medical care. Cooperation may be through the actual provision of services on a regular basis, accepting clinical referrals, or serving in an advisory capacity, or a combination of these.

Medical-Legal Page

Malpractice Not Found In Treatment of Cardiac Arrest

In an action for malpractice, physicians were entitled to have the phrase "unbroken by any new and independent cause" included in the definition of proximate cause in a jury instruction, a Texas appellate court ruled. The court found that there was ample evidence to support a conclusion that an agency unrelated to a physician's alleged negligence was the immediate cause of poor circulation in the area of a spinal cord injury at the time of cardiac arrest.

While a 42-year-old patient was undergoing a hysterectomy, she suffered a cardiac arrest. She was given emergency treatment, and her heart resumed beating an hour and 20 minutes later. The operation was resumed when the arrest ended. About an hour after the cardiac arrest was discovered, an electrocardiogram was performed.

Shortly after the operation, it was found that the patient had suffered a transverse myelitis of the spinal cord at about the level of the 11th thoracic vertebra and was paralyzed below the waist. She brought action for malpractice against three physicians and the hospital where the operation was performed. The jury brought in a verdict for two physicians and the hospital. It found that the third physician was negligent in failing to obtain an electrocardiogram sooner than he did and in failing to give the patient sodium bicarbonate sooner. However, the jury found that neither failure was a proximate cause of the patient's injuries.

On appeal, the patient complained of inclusion in the definition of proximate cause of the phrase "unbroken by any new and independent cause" and the definition of "new and independent cause." According to the definition given by the court, proximate cause meant a cause that in its "natural and continuous sequence, unbroken by any new and independent cause," produced a result that would not have happened without that cause. "New and independent cause" was defined as an act or omission of a separate agency that was not reasonably foreseeable and that destroyed the causal connection and became the immediate cause of an injury.

The patient objected to the definition of proximate cause on the grounds that it was prejudicial to her in that it indicated to the jury that there was a new and independent cause present and left the jury to speculate about a possible separate agency not raised by the evidence.

The appellate court found that there was ample evidence to support the jury's failure to find that the physician's negligence was a proximate cause of her injuries. The court pointed out that an electrocardiogram is only a gauge of cardiac activity and does not cure anything. There was expert testimony that since there was no permanent brain damage after the cardiac arrest, and since the brain is the organ most sensitive to an insufficient blood supply, sufficient oxygenated blood must have gotten to the various parts of the body during the cardiac arrest.

Expert testimony was also given that if during the cardiac arrest enough blood was pumped to avoid permanent brain damage and the heart was resuscitated, the arrest had been successfully treated and the poor circulation to the spinal cord must have been caused by something such as a plaque or a clot blocking the blood flow. There was testimony that the patient's myelitis was caused by an occlusion of the anterior spinal artery and might have occurred either during the cardiac arrest or shortly thereafter.

As to the patient's contention that the physician failed to give her sodium bicarbonate soon enough, two physicians testified that this was not a cause of the patient's injury. The court found that the testimony was sufficient to support a jury conclusion that there was enough oxygenated blood to avoid brain or spinal cord damage, as well as enough evidence to support a conclusion that an agency unrelated to negligence by the physician was the immediate cause of the poor circulation that caused the spinal cord injury. Affirming the judgment of the trial court, the appellate court found that the physicians were entitled to have the questioned phrase included in the definition of proximate cause.—*Hunter v. Robison,* 488 S.W.2d 555 (Tex.Ct. of Civil App., Oct. 26, 1972; rehearing denied, Nov. 22, 1972)

Negligence in Administration Of Anesthetic

In an action for malpractice, where evidence narrowed the probable causes of death to two factors, both of which were attributable to negligence by a nurse anesthetist, the Texas Supreme Court ruled that the husband of a deceased patient had a prima facie cause of action against the anesthetist.

The patient, a 30-year-old woman, was admitted

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to the hospital for repair of a diaphragmatic hernia. Thirteen minutes after the nurse anesthetist began administering the anesthetic, the patient died.

The patient's husband and minor children brought action for negligence against the anesthetist, the hospital, and two physicians. They alleged that the anesthetist was negligent in administering an improper mixture of halothane with oxygen and nitrous oxide, improper use of the anesthesia machine, and improper monitoring of the patient while she was receiving the anesthetic.

A physician testifying for the husband said that in reasonable medical probability the patient's cardiac arrest was caused by an overdose of halothane or lack of proper oxygenation. There was proof that the anesthesia machine was set so that the flow rate of the gas mixture was only 2 liters per minute instead of 4 liters, as it should have been. The lower flow rate would magnify the vaporization of halothane, a very potent drug, and intensify its strength.

When asked which anesthetic agent was given in an overdose, the physician said it was probably halothane. The physician also said that the machine setting was not according to the manufacturer's instructions and that compensation for the temperature factor, which caused the halothane concentration to vary from double or half that intended, was required. He said that a temperature variance of 2 degrees could change the drug concentration 7 per cent. The anesthetist testified that she could not calculate the halothane concentration to be administered and that she forgot about the effect of temperature.

The physician said that there were two possible causes of the patient's death. Either the patient was not getting enough oxygen or her cardiac arrest went unnoticed for three to five minutes. He said he knew of no other possible cause.

The husband proved that while the patient was being given the anesthetic, the anesthetist should have been taking blood pressure readings at two-minute intervals instead of every 3 or 4 minutes, as she had been doing. He said that blood pressure readings were the best indicators of the depth of anesthesia and that if the readings were too far apart the anesthetist could fall behind in assessing the effect of the anesthetic on the patient. He said that taking blood pressure readings at three to five minute intervals would make it possible for a cardiac arrest to occur at a time that was too remote for reversal of the arrest and resuscitation of the patient.

The nurse contended that the evidence did not establish causation because there was proof of other possible non-negligent causes of the patient's death and because the physician who testified failed to meet the community standard test as a predicate for his testimony.

The trial court dismissed the hospital from the case and entered judgment on an instructed verdict for the other parties being sued. On appeal, the court said that another possible cause of the patient's death was her allergic reaction to halothane. The physician who performed the autopsy said that this was a possible cause, but when directly questioned he said that there was no allergic reaction.

The appellate court found that the husband's appeal from the order dismissing the hospital was not timely, that his notice of appeal was inoperative as to the hospital, and that the instructed verdict for the other three parties was proper.

The state supreme court found that the trial court properly admitted the testimony of the physician who appeared for the husband. He recognized that procedures used by nurse anesthetists differed between nurses and anesthesiologists and in certain localities, but he said that there are certain minimum safe and accepted practices that could not vary because of uniform human tolerance to certain conditions. He said that the anesthetist's negligent acts fell below the minimum standards for her profession. The court held that the husband proved a prima facie cause of action against the nurse anesthetist.

The husband contended that he proved independent grounds of negligence as to the physicians' efforts to reverse the cardiac arrest. The physicians conceded that they were vicariously liable if liability were found on the part of persons under their supervision in the operating room. The court found it unnecessary to comment about the evidence on other points, holding that the cause must be reversed as to all the parties being sued and sent back to the trial court.—*Webb v. Jorns*, 488 S.W.2d 407 (Tex.-Sup.Ct., Nov. 15, 1972; rehearing denied, Jan. 10, 1973)

Editor's Note: A prior decision was reported in *The Citation*, Vol. 24, No. 10, p. 146.

Sibling Rivalry

(Continued from page 318)

time, the state journals *can* continue the attempt to increase the readership within their areas (and what area is not trying to get more physicians?). They can attempt to increase their readership by striving to improve the quality and appeal of their material (and what journal would not be pleased to do that?). And they can encourage their readers to express to the advertisers their appreciation of these facts of publication life (and what do you think this is?).—*D.E.G.*

Woman's Auxiliary

. . . *Annie Pays Tribute to "Doc's Girl"*

Annie thinks it's time she said something about some of the Auxiliary Annies who do double duty being physicians as well as wives of doctors. There's quite a few of these talented ladies around Kansas, but there's one in particular that Annie wants to pay particular attention at this time. And that's because she's really a very special person and an inspiration to all of us. Who is this wonder-girl? No one else but Irene Koeneke Hertzler . . . widow of the famous "Old Doc."

One might ask what makes Dr. Koeneke so special. There isn't room enough on this page to really tell all the reasons, but the main one is that along with being a successful and much-revered physician, she has "carried the torch" for the Hertzler Clinic and Old Doc.

A small, grandmotherly-looking woman with sparkling white hair curled and pulled up on top of her head, her blue eyes twinkling with humor at her reminiscences, Irene Koeneke's gentle manner and looks belie her innate efficiency and ability. Recalling the first time she saw Dr. Hertzler, when she was a medical student at the University of Kansas, Dr. Koeneke later told a classmate he was "the homeliest man I'd ever seen." Between her junior and senior years in medical school, she applied at the Hertzler Clinic for a summer externship as one of Hertzler's "cubs." One of the first people she met at the Clinic was "the Chief." When she was introduced to him, the doctor looked at her and said "Girl, we're glad you're here," and his eyes were the kindest she had ever seen. From that time on she never thought he was homely.

Most people thought Dr. Hertzler was a man of few words, but "he chattered like a magpie to me," Dr. Koeneke said. They started their day at 5:30 a.m., visiting while they ate breakfast, then continued their conversation in the evening after both had put in a day at the hospital. Because of these conversations, and their mutual contentment in each other's company, she states that she has never felt she was alone since the doctor's death. Knowing how Dr. Hertzler felt about so many things has made the monumental task of continuing his work at the clinic somewhat easier.

Following Dr. Hertzler's death, the town of Halstead asked that some kind of memorial be made to their beloved friend. Knowing her husband's love for children, and knowing how he had felt about the need for visual health education, Dr. Koeneke established the Hertzler Research Foundation and the Kansas Health Museum. The museum has grown so much that new quarters are being built this year on the city block that used to house the old Halstead High School. Lessons in nine different areas of health education are used. This includes a film, lecture, watching "Valeda," the transparent talking woman, and a time for browsing among the exhibits. On a busy day it isn't uncommon to have as many as 170 students go through the museum. This is particularly true during March, April, and May when schools take their field trips.

This spring also marked the fourth annual Hertzler Memorial Lecture. Each year papers of highest quality are presented by speakers to area physicians and their wives. This is only a part of the continuing education program offered by the Foundation. In 1970, a teacher training program was initiated with two hours of postgraduate credit given by Kansas University to the 22 teachers taking the two-week summer course in health education. Also, much of Dr. Hertzler's famous library has been rebuilt and any doctor may borrow its books.

Following Dr. Hertzler's death, Dr. Koeneke assumed the responsibility of the Hertzler Clinic. Its roster of physicians is most impressive. When asked how she felt about the responsibility of carrying on the work of such a famous clinic, Dr. Koeneke's only remark was a humble, "It hasn't been a responsibility. It has been a privilege."

Somehow one gets the feeling that the good doctor is looking down from somewhere with those blazing black eyes full of pride and saying "Girl, we're glad you're here."

Speaking for the Auxiliary and for your continued support of our efforts, we're glad too, Irene.

Auxiliary Annie

The Journal accepts short classified advertising from the members of the Kansas Medical Society without a charge. These ads run in three consecutive issues of the Journal and are keyed with a correspondence number. All replies are forwarded immediately to the advertiser. Other brief classified ads are accepted from members of the medical profession only upon approval of the Editor or Editorial Board.

The D. C. Line

Editor's Note: The Editorial Board feels that the Kansas Medical Society would profit by maintaining contact with Representative Bill Roy, both as a source and interpreter of political activities on the national medical front. Accordingly, we are initiating a monthly column written by Dr. Roy. It is hoped that this will not only provide the service intended, but invite a response of readership either through the JOURNAL or to Dr. Roy directly.

In this column, I will discuss the present status of —and the prospects for—national health insurance (NHI) legislation. Next month, I will deal with the substance of a bill which I expect to have introduced by that time.

Comprehensive NHI legislation can best be characterized as dormant at this time. This is the result of several factors. First, the Ways and Means Committee of the House is completely occupied with the consideration of two other extremely important pieces of legislation—tax reform and tariff and trade legislation.

Secondly, the Nixon Administration has not placed any NHI legislation before this Congress. The legislation of the last Congress, the National Health Insurance Standards Act and Family Health Insurance Program, have been reintroduced by members, but these programs no longer have the support of the Administration. The Department of Health, Education and Welfare, and the influential Office of Management and Budget have NHI under consideration, and it has been said that legislation will be forthcoming this fall.

There are indications, however, that the Administration proposal may be further delayed by the present difficulty in policy-making resulting from the attention-consuming Watergate hearings.

A third and very good reason that NHI consideration is lagging is the fact of large federal budget deficits. NHI does not have to be financed out of the general funds of the federal government or by money from the Social Security Trust Fund. But many proposals call for such financing, and the unavailability of such funds restrains the consideration of this kind of NHI legislation.

One recent development has renewed interest in NHI. This is the prospect that our Health Subcommittee will hold NHI hearings late this year. Our hearings would also be limited by time available. A one-year renewal of twelve titles of the Public Health Service Act was recently signed into law. We have properly committed ourselves to a thorough review of this Act and the rewriting, reform, or discontinuation of the programs under this Act between now and June 1974.

This will be time-consuming, because included in this Act are comprehensive health planning, regional medical programs, hospital construction support, family planning, and a number of other important categorical programs.

Any NHI hearings before our Subcommittee will also be limited by jurisdiction. We cannot legislate definitely regarding financing from the Social Security Trust Funds or Medicaid (Title 19 of the Social Security Act). We do have jurisdiction over private health insurance and we can consider the availability and scope of health insurance for our citizens.

Because of my conviction that we should not pay out of the general funds of the federal government or the Social Security Trust Funds for health care services for those who can afford to pay for their own health care, I have been working for the last eight months on a national health insurance bill which hopefully will be referred to our Health Subcommittee. This bill, to be introduced this summer, will probably be the basis for any hearings held by our committee. I will discuss the content of the bill next month.—*W.R.R.*

Journal on Microfilm

Microfilmed copies of current as well as all back issues of the JOURNAL are available through University Microfilm Services, a subsidiary of Xerox Corporation. The 35 mm film fits all standard viewers and provides the JOURNAL in miniature at a savings on binding and storage costs. Write for information or send orders direct to University Microfilm Services, 300 North Zeeb Road, Ann Arbor, Michigan 48106.

Personalities—IN KANSAS MEDICINE

H. L. Patterson, Larned, is discontinuing his practice and will be moving to Pakistan.

Earl V. Carlson and **Glen C. Hutchinson,** Hays, participated as instructors, medical coordinators, and advisors in a basic training program for Emergency Medical Technicians for Northwest Kansas.

A book based on the life of **Charles L. Youngman,** Harveyville, who practiced medicine here for 70 years, has gone to print. Those interested in obtaining a copy, should contact Paul Youngman, Rt. 2, Box 127, McMinnville, Oregon 97128.

Andres Grisolia, Leavenworth, was appointed associate clinical professor of surgery in the department of orthopedic surgery, KUMC.

John F. Lance, Jr. and **H. O. Marsh,** Wichita, participated in the filming of "A New Kind of Joint." The film was prepared by the Arthritis Foundation, and shown on KPTS-TV, Channel 8, Wichita.

Joseph L. Kyner, formerly of Wilson and now on the staff of KUMC, has written a historical article which appeared in the April issue of *JAMA.*

C. E. Brown, Stafford, attended a medical conference in Denver.

The 1973-74 Boss of the Year of the area American Business Women's Association is **Jack E. Lungstrum,** Salina.

DeWitt S. Lowe, Hiawatha, participated in the Northeast Kansas Senior Citizens activities recently.

D. B. Foster, Topeka, collaborated on a clinical report which was presented at a joint meeting of the American Neurological Association and the Canadian Congress of Neurological Sciences, in Montreal.

Kenneth W. Carbaugh, Mission, retired after practicing medicine for 41 years in this area.

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Vox Dox

Vox Dox Editor:

Again, this year I am compiling case reports of allergic reactions to biting insects, i.e., mosquitos, fleas, gnats, kissing bugs, bedbugs, chiggers, black flies, horseflies, sandflies, deerflies, etc. I am also interested in reactions to the Imported and Southern Fire Ants.

I would like physicians to supply me with case reports of those patients who have had reactions to such insects. Include in your reports, the type of reaction and the complications, if any, the age, sex, and race of the patient, the site of the bites, the season of the year, the immediate symptoms, the skin test results, desensitization results, if any, and any associated other allergies. Send this information to the following address: Claude A. Frazier, M.D., 4-C Doctor's Park, Asheville, N. C. 28801.

Thanking you in advance, I remain

Sincerely,
CLAUDE A. FRAZIER, M.D.

Williams-Steiger Act

(Continued from page 315)

our sincere intention to be as reasonable as possible in administering the Act.

To whatever degree the allied medical fields can improve occupational safety and health through preventive measures, physicians may be able to focus significantly more of their efforts toward a number of health problems which, even now, are not beyond their expertise but merely outside of the time they can devote to such problems. The assault upon human suffering seems one of the few goals common to the whole of mankind. Although some tangible value can be more easily placed upon the elimination of suffering once it is felt than upon its prevention, the latter still seems to justify considerable public and individual effort.

Acknowledgement

Richard Shanteau, Robert Borchardt, and Patricia Adams for their comments.

References

Available from the authors.

Vox Dox Editor:

The following is a letter I wrote to Dr. Jack D. Walker and Dr. Lawrence Perry, in charge of the General Practitioner course being organized or facilitated at KUMC:

"I have been told I am a 'sick man' by some of my colleagues over the state of Kansas because of my attitude toward abortion, the alcohol-drug problem, the venereal disease-pornography problem, and the peer review problem.

"The article in the *Kansas City Star* on July 12, 1973, entitled 'General Medicine Growing,' gave me a severe relapse to my illness. I had severe nausea.

"The causes for this severe nausea were these quotes:

" 'This program offers three years more of education. It trains the doctor in all the things he'll be required to do as a family doctor.'

" 'Sitting next to Dr. Perry's desk at the clinic was a *black bag,* once the symbol of a physician. The image of a doctor with a black bag filled with his equipment is the image centers like the K.U. Medical Center is trying to promote. "Family practice is 80 per cent of the practice," Dr. Perry said. "Specialists have to have the sophistication of a hospital backing them. But a general practitioner needs to have all his tools with him. He can't afford to depend on a hospital." '

"The nausea overwhelmed me when you suggested *three more* years of education to prepare these young men to do the things the specialist never can do, and that is to stop in once a week to see an incurable cancer patient. 'It may not help him medically, but if it makes him feel a little better then that's what we're here for.' And to think of *three more years of education* to do this sort of thing. Hand me that emesis basin again."

Sincerely,
A. S. REECE, M.D.

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

Book REVIEWS

PRACTICAL AUTOMATION FOR THE CLINICAL LABORATORY edited by Wilma L. White, Marilyn M. Erickson and Sue C. Stevens. The C. V. Mosby Company, St. Louis. 1972. 591 pages, 346 illustrations. $22.50.

This book should appeal to the medical directors and technical personnel of any moderate to large clinical laboratory. The editors and contributors have described many of the automated and semi-automated instruments, now available for use in clinical laboratories.

This book begins with a brief introduction to the general aspects of automation, then follows up with a very good forty-four page review of basic mechanical and electrical physics and applies it to common laboratory equipment, as examples. This section contains some excellent tips on safety, necessary tools and general trouble shooting procedures.

The next six sections deal with automated equipment and instruments in each of the clinical areas: Chemistry, blood bank, serology, microbiology, hematology and cytology. In each section, the various individual instruments are described; the operation and general principles, the specific components and their function, operation, maintenance, trouble shooting and available service.

Notable additions to this edition have been descriptions of Beckman's DSA 560, Electro-Nucleonic's GEMSAEC, Hycel's Mark X and Instrumentation Laboratories' Clinicord System.

The chapters on AutoAnalyzer modules and chemistry have been expanded and brought up-to-date, and a chapter on upgrading older AutoAnalyzers added.

There has been no attempt to evaluate, or compare, these instruments and each instrument has been described by personnel who are either currently using them, or involved in marketing from the manufacturer of the instrument, so some bias can be expected.

The editors finish with a short section on computer technology and a new and fairly complete glossary.

Clinical laboratory instrumentation is a very rapidly developing field and any published information is soon obsolescent. Yet, even with this in mind, this book would be a good investment for any clinical laboratory, or teaching program for medical technology.—*A.A.F.*

MALNUTRITION, ITS CAUSATION AND CONTROL, Vol. I & II by John R. K. Robson. New York, Gordon and Breach. 1972. 613 pages, illustrated.

This book of two volumes is an attempt to bring together the various facets of malnutrition. These two volumes uniquely do this in the fact that they show how there are many facets that have not been considered. The ones that we thought were unusual and bear much consideration is the fact that malnutrition is tied up with social, cultural, environmental and economic factors. At the same time, they point out that even though there may be an abundance of a particular food product available, this might not be used in the dietary process either by reason of custom, habit or prejudice. In evaluating how to correct malnutrition, therefore, we must take into consideration that some places will need a food supplied to make up a malnutrition state but in other places, the food may be available and we will need to teach them either how to use these foods to the greatest advantage or gradually work to overcome prejudice, custom or cultural outlooks. The book in our opinion, has done a very extensive work and challenges us to look at malnutrition from a more broad viewpoint than just what the patient looks like to us.—*C.A.N.*

INTRODUCTION TO NEUROSCIENCE by Jeff Minckler, M.D., Ph.D., Editor. The C. V. Mosby Company, St. Louis, 1972. 420 pages, 661 illustrations. $22.50.

This is an outstanding introductory textbook on the nervous system. Major attention is directed to the gross anatomy, histology, embryology and physiology of the nervous system, with helpful sections on electron microscopy, neurochemistry, neuroradiology and nervous system tissue culture. The drawings and diagrams are superior. The emphasis throughout is on integration of the basic sciences as they relate to the structure and function of the central and peripheral nervous system. The book would be useful to medical, veterinary and dental students, neurologic technicians (as in electroencephalography, electronystagmography, radiology), physical therapists, and psychologists.—*D.B.F.*

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VOL. LXXIV
NO. IX

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Contents for September

Scientific Articles

Thoracic Outlet Relief: First Rib Excision—W. D. Hoofer, M.D. and A. Dean Burnett, M.D., Halstead . . . 329

Splenic Cysts: Epidermoid Cyst of the Spleen—J. W. Graves, M.D. and A. K. Tayiem, M.D., Wichita . . . 332

Functional Obstructive Cardiomyopathy: Familial Idiopathic Hypertrophic Subaortic Stenosis in Children—Timothy K. Burch, M.D. and Eugene F. Luckstead, M.D., Kansas City, Kansas . . . 335

Medical History

Joseph Brenneman, 1872-1944—Ronald D. Greenwood, M.D., Boston, Mass. . . . 339

Socio-Economics

Disability Insurance Under Social Security—Dwight Lawson, M.D., Topeka . . . 343

Miscellaneous

Advertisers . . . 18
AMA Committee on Community Health Care . . . 353
AMA House of Delegates Report . . . 349
Announcements . . . 359
Book Reviews . . . 358
Classified Advertisements . . . 18
Editorial Comment . . . 346
Influenza Vaccine Recommendations . . . 360
Information for Authors . . . 360
KMS Committee Structure . . . 356
KUMC Postgraduate Education . . . 14
Medical-Legal Page . . . 351
New Members . . . 355
Obituaries . . . 348
Personalities in Medicine . . . 355
President's Message . . . 345
Vox Dox . . . 352

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Scientific ARTICLES

Thoracic Outlet Relief

First Rib Excision

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RELIEF OF NEUROVASCULAR compression by first rib resection was reported by Murphy[1] in 1910. Little attention was given to this mode of treatment until Falconer and Li,[2] and Clagett[3] each reported success with first rib resection. Until the term "thoracic outlet syndrome" was advanced by Rob and Sandever[4] in 1958, upper extremity neurovascular compression was divided into the scalenus anticus syndrome, hyperabduction syndrome, costoclavicular syndrome, cervical rib syndrome, cervicobrachial compression syndrome, effort vein thrombosis, subcoracoid pectoralis minor compression, and first thoracic rib syndrome.[5]

Each was diagnosed by a special physical maneuver, such as Adson[6] described for the scalene compression, but as pointed out by McBurney and Howard,[7] the common factor was the first rib.

Anatomy

Anatomically, the thoracic outlet is bounded by the first rib inferiorly, over which the vessels and nerves must ascend before descending into the arm. Anteriorly and superiorly is the clavicle and anterior scalene muscle. The medial scalene muscle is posteriorly. Laterally, the vessels and nerves must pass beneath the pectoralis minor tendon. Thus, with the vessels and nerves of the upper extremity drawn tautly over the first rib, with contraction of the anterior scalene muscles and with posterior travel of the clavicle, the neurovascular bundle is imprisoned in an anatomic scissors, causing neural compression and obliteration of the arterial and venous flow. Elevation of the arm brings the structures against the pectoralis minor tendon and coracoid process with similar results.

Symptoms

Thoracic outlet compression symptoms may be divided into vascular and neural. Vascular compression symptoms may be either venous, arterial, or both.[8] Venous compression will cause engorgement of the extremity and may cause hand and finger edema. Due to chronic stasis, axillary vein thrombosis is not infrequent. Arterial insufficiency from subclavian compression causes the extremity to be cold, pallid, with tingling, fatigue, and occasional muscle cramps. Severe insufficiency, claudication, and Raynaud's phenomenon may be associated with compression.

One may consider removal of a rib as the first operation performed on man. Controversy continues as to the wisdom of the original procedure, as well as in resection of the first rib in the treatment of thoracic outlet compressive syndromes.

By far the most disabling, however, is neural compression. Compression of the brachial plexus causes pain, numbness, dysesthesia, weakness, heaviness and clumsiness of the extremity. The pain may radiate from the area of compression up the back of the neck, out to the point of the shoulder, and along the medial edge of the scapula. Due to radiation into the upper anterior chest, the pain is occasionally con-

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fused with angina pectoris. Dysesthesia may radiate into the hand and arm in an unusual pattern, but is usually confined to the dermatome subtended by the ulnar and, less often, the median nerves. The extremity is usually weaker than its mate. Clumsiness due to incoordination frequently is a presenting complaint. Severe, long-standing compression will lead to interosseous and thenar atrophy.

The arm tires quickly and feels heavy. Working the extremity in an elevated position, as in combing hair, holding a steering wheel, or painting commonly aggravates the symptoms. Many complain of the arms and hands "going to sleep" at night, and of having to arise and shake the hands and arms to gain temporary relief.

Many of the early symptoms are general in nature, and physicians not familiar with this syndrome may consider the symptoms to be on a functional basis.

Diagnosis

The diagnostic signs of thoracic outlet compression are both vascular and neurologic.

Vascular compression may be demonstrated by placing the arm in 90° of abduction[1] with the shoulders "squared" in military fashion. The patient must be placed at ease and relaxed,[8] since a tense patient can elevate the clavicle and prevent its usual scissors action at the first rib level. With the arm in the abduction position and extended, it is placed in external rotation, palpating the radial pulse simultaneously. Frequently, the pulse will be obliterated and, by rotating the arm in and out of external rotation, the pulse may be "turned on and off," much like a water faucet. Many patients will complain of their usual symptoms during this maneuver. If they are asked to open and close the hand rapidly in this position, claudication of the forearm muscles begins, which is relieved by taking the arm out of external rotation. In some patients, the classical "Adson test" will be positive, however, with extensive use it has been found to be totally unreliable.

Neurologic findings are usually related to either the median or ulnar nerves, the latter being by far the most frequent. Sensory examination demonstrates hypesthesia over the dermatome subtended by the particular nerve. Rarely, an early case of compression will cause irritation and hyperesthesia in that dermatome. Interosseous muscle atrophy is frequently seen with ulnar compression, but thenar atrophy is rarely seen with median nerve compression. One must beware of the latter, since thenar atrophy is more frequently seen with carpal tunnel median nerve compression. In older patients, it is not unusual to have both thoracic outlet and cervical osteoarthritic neural compression simultaneously. In these patients, one must rely on the degree of vascular signs as well as on nerve loss.

The differential diagnosis must include trauma, ruptured cervical disc, cervical arthritis, spinal cord tumor, tendinitis, capsulitis, shoulder arthritis, myositis, angina pectoris, ulnar nerve entrapment at the elbow, and the carpal tunnel syndrome. Most of these can be ruled out, since none will cause both vascular and neural symptoms specifically related to position and use of the arm.

Occasionally, difficulty in separating the above causes of pain may require nerve conduction studies or arteriography.[9] This has not been necessary in our experience.

Material

In 1970, a prospective study was initiated at the Hertzler Clinic to evaluate the results of first rib resection for the thoracic outlet syndrome. To qualify for this form of treatment, each patient had to be demonstrated to have both vascular and nerve compression with a marked nerve loss. Each patient was examined by one investigator, who either scheduled or declined surgery. All operative procedures were performed by the same investigator with the same technique. Each patient was re-examined during the postoperative period, after one month, three months, and approximately 80 per cent of all patients after one year.

A total of 133 patients qualified for these treatment criteria. However, two additional patients were in-

TABLE 1

ANALYSIS OF 135 PATIENTS WITH THORACIC OUTLET SYNDROME

	Number
Female	105
Male	30
Symptom	
Pain	134
Numbness	133
Weakness	128
Incoordination	128
Claudication	135
Raynaud's phenomenon (unilateral)	4
Edema	1
Extremity	
Bilateral	97
Right	21
Left	17
Nerve Involved	
Ulnar	108
Median	21
Combined	6
Cervical ribs	5
Carpal tunnel syndrome	2
Duration of symptoms (average, months)	17.4

cluded due to severe vascular compression, with vascular complications from this compression.

Technique

The operative technique is simple and standardized. The patient is placed in the lateral decubitus position with the padded arm elevated and attached to the ether screen. The anesthetist palpates the pulse in this arm throughout the procedure to prevent inadvertent injury to the neurovascular bundle by overzealous retraction. A curving incision is made below the hairline of the axilla, deepened to the chest wall, and the axillary fat pad and neurovascular bundle are elevated for visualization of the first rib. Effort is made to preserve the intercostal brachial sensory nerve; however, this is not always possible. The periosteum of the first rib is incised and completely stripped, thus transecting the insertion of the scalene musculature. Care must be taken on the underside of the rib, due to the thinness of the periosteum, endothoracic fascia and pleura, to avoid entering the chest cavity. After periosteal mobilization, the rib is excised to the level of cartilage anteriorly, and posteriorly to the transverse process. The quality of decompression is obvious by descent of the neurovascular bundle and the bounding pulse after the rib resection. The wound is closed by approximation of the axillary fat pad and skin.

Results

Of the 133 patients who underwent transaxillary rib resection for neurovascular compression, and two patients who were treated for severe vascular compression *(Table 1)*, 105 patients were female and 30 male. The age ranged from 22 to 74, with the average of 43. One hundred thirty-four patients complained of pain, 133 of numbness, and 128 of weakness and incoordination. The duration of symptoms ranged from one month to 13 years, the average being 17.4 months. Symptoms were bilateral in 97 patients, on the right in 21, and on the left in 17. The ulnar nerve was involved in 108 patients, median nerve in 21 patients, and combined median and ulnar nerve in 6 patients. Two patients had a concomitant carpal tunnel syndrome. Five patients had cervical ribs. These were resected with the first rib. Twenty of the 97 patients with bilateral compression required bilateral rib resection, and 77 had unilateral rib resection.

All patients had return of nerve function following operation. The two patients without nerve loss, but with vascular compression, had relief of their vascular symptoms.

In many instances, return of nerve function was immediate when preoperatively only hypesthesia was present. Those with muscle atrophy averaged three months before muscle growth occurred. Return of interosseous muscle strength was highly variable, and usually was proportional to the patient's eagerness in following the hand exercise regimen. There was no mortality in this group of patients. Fifteen complications of their illness were listed, eight of which were a result of operation *(Table 2)*.

Two of the four patients with Raynaud's phenomenon required thoracic sympathectomy for control of vascular constriction, while two had full relief with rib resection. The patient with postoperative axillary artery thrombosis was fully relieved by Fogarty catheter extraction of thrombus, under local anesthesia approximately one hour after the rib resection.

Approximately 60 per cent had axillary numbness postoperatively due to intercostal brachial nerve section or stretching. In approximately 20 per cent this was permanent, but not disabling. Women were more greatly affected than men, due to their usual custom of shaving the underarms.

TABLE 2

COMPLICATIONS

	Number
Preoperative	
Raynaud's phenomenon	4
Axillary vein thrombosis	1
Concomitant	
Carpal tunnel syndrome	2
Operative	
Pneumothorax	1
Axillary artery thrombosis	1
Hematoma or seroma	3
Hyperesthesia (ulnar)	3
Total	15

Comment

The average patient seen during this investigation was a 40-year-old female complaining of shoulder, neck, and arm discomfort and of her arms going to sleep at night, or when working in an elevated or reaching position. Using the criteria requiring demonstrable vascular compression and nerve loss, only one of every five patients seen for evaluation was scheduled for surgery.

It is the authors' opinion that most patients with only vascular compression can be relieved nonsurgically by changes in position, weight loss, teaching of elevation of the shoulders while sitting, muscle relaxation, and mild sedation. If surgical therapy is confined to those patients with demonstrable nerve

(Continued on page 352)

Splenic Cysts

Epidermoid Cyst of the Spleen

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SINCE THE FIRST REPORT[1] of epidermoid cysts of the spleen in 1929, there have been 49 cases reported. Dibble and Weigent reviewed the 44 cases known before 1965.[2] The fiftieth case in the literature of epidermoid cyst of the spleen is reported in this paper.

Case History

A 25-year-old white female was admitted to St. Francis Hospital on April 23, 1972, complaining of pain in the left upper abdomen which started a few days earlier while she was arranging her furniture. A palpable mass was felt extending four fingerbreadths below the left costal margin. The mass moved with respiration.

X-ray examination showed a left upper quadrant mass displacing the left kidney inferiorly *(Figure 1)*. Liver scan was normal, and splenic scan showed splenomegaly with a large defect in radioactivity *(Figure 2)*.

The hemogram, urine analysis, liver, and renal function tests were normal. A bone marrow examination was normal. Immuno-globulin electrophoresis was normal and antinuclear antibody was absent. Cultures of the blood and the stool were negative.

The spleen was removed on April 27, 1972, using an upper-midline incision which was extended to a thoraco-abdominal incision in the seventh intercostal space. The left pleural cavity was drained by two chest tubes, and the abdomen by two Shirley's tubes. Postoperative course was uneventful and the patient was dismissed on May 7, 1972.

Pathologic Examination

The specimen consisted of spleen measuring 24 × 26 × 8 cm containing a cyst measuring 16 × 10 × 5 cm, which had a wall thickness of 0.5 cm *(Figure 3)*. The cyst contained 2,500 cc of brownish-gray fluid with cholesterol crystals, and the inner surface of the cyst was trabeculated and yellowish-gray in color.

Microscopic examination showed the cyst to be lined with stratified squamous epithelium without keratinization. The wall was composed of dense hyaline collagenous tissue with granulomatous formation in some areas. Splenic parenchyma was normal, but compressed and showed areas of fibrosis and moderate congestion *(Figure 4)*. The pathologic diagnosis was epidermoid cyst of the spleen.

Discussion

Splenic cysts of various types are not common. Haffner[3] reviewed 563 cases prior to 1966.

Splenic cysts are classified into primary or true cysts, in which the cyst wall is composed of epithelial or endothelial tissue, and secondary or false cysts, in which the cyst has no distinct wall[1] *(Table 1)*.

Among neoplastic cysts, 40 per cent are hemangiomas and 20 per cent are epidermoid cysts.[4] Parasitic cysts comprise about 3 per cent. Most splenic

Figure 1. IVP. Notice left kidney displaced inferiorly by left upper quadrant mass.

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TABLE 1

CLASSIFICATION OF SPLENIC CYSTS

I. Primary or true
  A) Congenital
  B) Traumatic
  C) Inflammatory
    1. Infoliation
    2. Dilatation
  D) Neoplastic
    1. Epidermoid
    2. Dermoid
    3. Lymphangioma
    4. Hemangioma
  E) Parasitic
II. Secondary or false
  A) Traumatic blood, serous
  B) Degenerative infarction, liquefaction
  C) Inflammatory T.B., necrosis

cysts are solitary. One case of multiple epidermoid cysts has been reported.[5]

The cause for epidermoid cysts is obscure *(Table 2)*. The average age of the patient is 16.3 years. Females are affected more than males (1.3:1).[6]

The clinical picture of splenic epidermoid cyst is variable. Coleman[6] reviewed 30 cases and found that 40 per cent were asymptomatic. Left upper quadrant pain was present in 30 per cent. History of trauma was found in 23 per cent of patients. On examination, 37 per cent of cases had palpable mass. Duration of symptoms varies from a few days to several years.[7] Two cases of epidermoid cyst have presented with signs of acute abdominal inflammation.[8, 9] One case presented with spontaneous rupture and massive hemoperitoneum.[10]

Figure 3. Notice the lower pole of the spleen at the right corner of the picture. Notice areas of fibrosis and thickening on the outer surface.

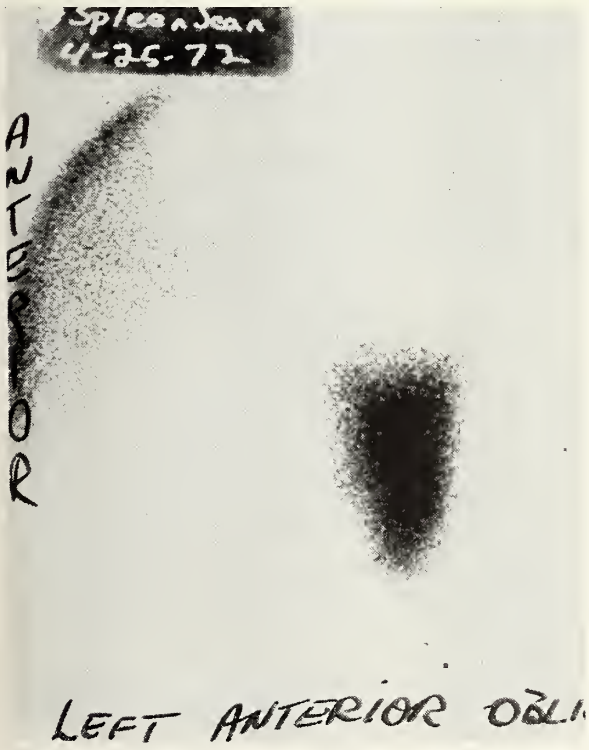

Figure 2. Splenic scan. Notice the defect in radioactivity in an enlarged spleen.

TABLE 2

ETIOLOGY OF EPIDERMOID CYST OF THE SPLEEN

1. Embryologic displacement (Shawan)
2. Autochthonous formation (Schneider)
3. Displacement of wolffian body and metaplasia (Santy)
4. Embryologic anomalies arising from mesodermal cell (Linn and Ellias)
5. Endodermal inclusion cyst metaplasia (Harding)
6. Metaplasia from infection, trauma, hemorrhage (Posener)

Figure 4. Section through the wall of the cyst. *a)* Stratified squamous epithelium lining of the cyst. *b)* Dense hyaline connective tissue. *c)* Compressed splenic tissue showing congestion.

Preoperative diagnosis is aided by radiologic signs of splenic enlargement in an otherwise healthy individual. Splenography, reported by Mossberg in 1966, aided in establishing the diagnosis of the cyst.

Splenic arteriography helps to differentiate between a cyst and other causes of splenomegaly.[11] Demonstration of a defect in radioactivity on splenic scan is probably the most expedient test available for demonstrating splenic cyst.[12]

The cysts vary in size, occupy usually one part of the spleen, and the rest of the splenic tissue is compressed. The outer surface is usually smooth but fibrotic; scarring and areas of infection are reported. Cholesterol crystals are found in many cases.[4]

The cyst is lined by stratified squamous epithelium which is characterized by:

1. Several layers of stratified squamous epithelium.
2. Prickle cells.
3. Intracellular bridges.
4. Absence of keratinization and epidermal appendages.

Keratinization has been reported by Lippit[9] and Mahour *et al.*[5]

The treatment for this condition is splenectomy; good exposure is warranted. Thoraco-abdominal incision is advised in very large cysts with diffuse adhesions.

Summary

We present a case of a 25-year-old female with epidermoid cyst of the spleen. Splenectomy was performed through a thoraco-abdominal incision. A brief review of the literature is included.

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Functional Obstructive Cardiomyopathy

Familial Idiopathic Hypertrophic Subaortic Stenosis in Children

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SINCE FUNCTIONAL obstructive cardiomyopathy was first described by Brock in 1959,[1] many reports have appeared in the literature. Once thought to be a rare entity, it is gradually being recognized as a distinct and not uncommon form of heart disease with synonyms such as: asymmetrical cardiac hypertrophy, hypertrophic obstructive cardiomyopathy, and idiopathic hypertrophic subaortic stenosis (IHSS).[2-4]

Two forms, familial and sporadic, have been described; most reports[2, 3] have been concerned with adults. The scattered reports in children usually present an asymptomatic child with typical clinical, radiographic, and electrocardiographic findings.[5] Recently, it has been suggested that familial cardiomyopathy may be a single entity with obstructive hypertrophy being one form of presentation.[6] This paper presents two brothers with familial idiopathic hypertrophic subaortic stenosis who demonstrate the variable clinical presentation and course of this entity. Extreme variability in the early clinical presentation of this cardiac abnormality deters proper identification of such patients. The role of beta-adrenergic blockade by Propranolol is discussed relative to its role in the medical management of this cardiac abnormality. Rapid cardiac deterioration ending in sudden death in one case and similar progressive changes in the other sibling at a later age characterize the clinical course in these two patients.

Case Reports

Patient One initially had a heart murmur detected at six months of age. Birth, growth, and development were within normal limits (20th percentile for height and weight). The murmur was again noted with chest x-ray and EKG abnormalities at eight months of age during hospitalization for pneumonia. At 18 months of age physical examination was unremarkable except for the cardiac findings. Cardiac examination revealed a mild generalized heave and no palpable thrills. Heart sounds were normal except for a slightly accentuated pulmonic second sound. A grade III/VI harsh, holosystolic murmur was heard maximally at the left lower sternal border. The peripheral pulses were bounding and blood pressure was 110/70 mm Hg. Chest x-ray revealed mild generalized cardiomegaly with normal pulmonary vascularity. The EKG demonstrated right axis deviation and biventricular hypertrophy with abnormal Q waves in the right precordial leads III, AVR, and AVF *(Figure 1)*; vectorcardiogram confirmed the EKG findings. Cardiac catheterization was performed at that time *(Table 1)*. Since the initial examination, the child's murmur changed to a grade III/VI systolic ejection murmur heard maximally at the left upper sternal border. Repeat cardiac catheterization at 5 years of age was essentially unchanged *(Table 1)*. At 6 years of age, the patient had a syncopal episode associated with a documented tachycardia requiring cardiac resuscitation including cardioversion. Following this episode, the child was placed on 40 mg Propranolol daily. Sudden death occurred at the age of 7 years while the patient was attending school.

The clinical, electrocardiographic, hemodynamic, and angiographic features of two brothers with a familial form of idiopathic hypertrophic subaortic stenosis (IHSS) are reported.

Patient Two was initially seen at 4 years of age. Birth, growth, and development had been normal (50th percentile for height and weight). This patient had recurrent hospitalizations for pneumonia, and on one occasion cardiac abnormalities in his chest x-ray and EKG were noted. No history of a cardiac murmur was obtained. The physical examination was unremarkable except for the cardiac findings. Cardiac examination revealed a mild left ventricular lift without a palpable thrill and a prominent apical third heart sound. A grade III/VI harsh systolic ejection murmur at the left upper sternal border, a high-pitched grade II/VI holosystolic murmur at

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TABLE 1

CARDIAC CATHETERIZATION DATA

Case	*Cath.*	*Age*	*RA*	*RV (apex)*	*RV (outflow)*	*MPA*	*P.A. (w)*	*LV (apex)*	*LV (outflow)*	*Ao*
1a	1	(1½ yrs.)	59	59	59	59				99
(O_2 sat. %)	2	(5 yrs.)	71	66	66	69		93		93
1b	1	(1½ yrs.)	(4)* a = 9	40/10	26/15	26/15		100/18**		100/44**
(Pressure; mm Hg)	2	(5 yrs.)	(5)* a = 12	60/10	20/10	20/10	(16)*	100/16**		100/60**
2a	1	(4 yrs.)	70	69	69	69				96
(O_2 sat. %)	2	(8 yrs.)	74	74	74	72	95			93
	3	(11 yrs.)	74	73	73	70				95
2b	1	(4 yrs.)	(1)* a = 5	30/6	30/6	30/10		110/18**		110/76**
(Pressure;	2	(8 yrs.)	(3)* a = 6	40/6	30/6	30/10	(18)*	140/18	120/18	120/55
mm Hg)	3	(11 yrs.)	(2) a = 8	60/6	30/6	30/10	(14)*	140/8	100/8	100/60

* Mean pressure
** Multiple hole catheter

the left lower sternal border, and a grade I/VI mid-diastolic flow rumble at the apex were noted on cardiac auscultation. Blood pressure was 90/60 mm Hg, and the peripheral pulses were bounding in quality. Chest x-ray revealed only mild left ventricular enlargement. The EKG demonstrated left axis deviation and mild left ventricular hypertrophy *(Figure 1)*. Cardiac catheterization was initially performed at 4 years of age *(Table 1)*. Clinical findings have remained stable. Repeat cardiac catheterization and angiograms at 8 and 11 years of age demonstrated mild mitral valve insufficiency and septal prominence *(Table 1) (Figure 2)*. Progressive subpulmonic obstruction (similar to Patient One) and subaortic obstruction were shown by serial cardiac catheterizations. Echocardiogram was diagnostic for hypertrophic cardiomyopathy. He is now receiving 40 mg of Propranolol daily.

Family History

The 32-year-old mother has had exertional dyspnea for 15 years, and chest pain on exertion on three separate occasions. Cardiac catheterization, echocardiogram, and clinical findings are consistent with the diagnosis of IHSS. The 38-year-old father had no clinical findings suggestive of heart disease. Two sisters, ages 12 and 3½ years, appeared clinically free of heart disease. None of the paternal relatives had histories suggestive of familial IHSS; a maternal uncle had cardiac catheterization and surgically confirmed IHSS.

Discussion

Although present diagnostic methods can confirm the diagnosis of IHSS, the pediatrician must first evaluate the child clinically before further diagnostic techniques are considered. The common types of functional murmurs should be excluded before the diagnostic possibilities of acquired or congenital

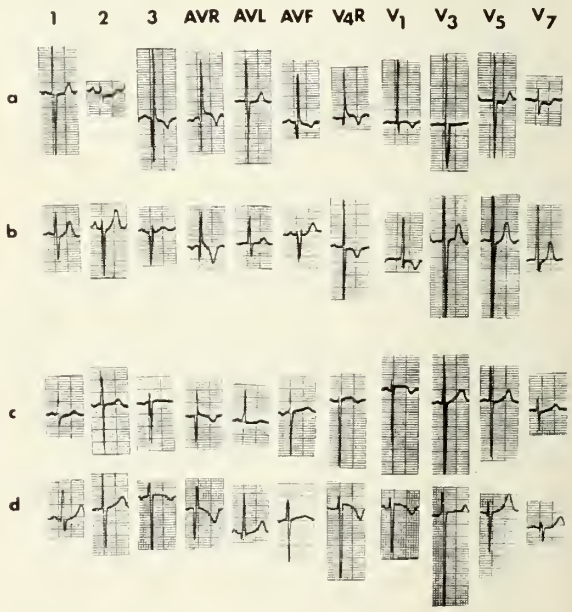

Figure 1. Standard electrocardiogram: (a) Patient One, at 18 mos. of age, (b) Patient One, at 5 yrs., (c) Patient Two, at 4 yrs. of age, (d) Patient Two, at 11 yrs.

heart disease are considered. The clinical diagnosis of idiopathic hypertrophic subaortic stenosis (IHSS) has subtle physical, radiographic, and electrocardiographic findings.

On physical examination, there is usually an accentuated apical left ventricular impulse described as a mild left ventricular "rock."[5] The systolic murmur is of varying quality, but most often described as a systolic ejection murmur at the left lower sternal border. This murmur can be accentuated by the Valsalva maneuver (forced expiration against a closed glottis). Less commonly, a holosystolic murmur is heard in the right upper sternal border.[5] The heart sounds are normal and systolic ejection clicks are notably absent.[7, 8] When the mitral valve is distorted by septal hypertrophy, one may hear the apical high pitched holosystolic murmur of mitral regurgitation.[9] An accentuated third heart sound is heard in approximately half the cases. Although peripheral pulses are generally not bifid, they are typically increased or prominent. A bifid arterial pulse contour is found in about 50 per cent of patients of IHSS.[2, 3, 10] Abnormal x-ray findings are found in about 80 per cent of the cases with the most frequent finding being left ventricular enlargement.[3]

EKG findings in IHSS are quite variable. The rhythm is usually sinus with a normal electrical axis in 60 per cent, left axis deviation in 30 per cent, right axis deviation in 2 per cent, and indeterminant axis in 7 per cent.[3] There is a higher incidence of right axis deviation and indeterminant axis in patients with familial IHSS.[7, 11] Abnormal Q waves are present in the precordial leads in approximately 50 per cent of the cases; these are thought to be manifestations of septal hypertrophy.[12, 13] The predominant EKG finding is left ventricular hypertrophy. Several series report EKG findings fulfilling one or more criteria of left ventricular hypertrophy in 70 per cent of the patients.[2, 3, 5] Progressive increase in prominence of Q waves were seen in both of these cases *(Figure 1)*.

The primary hemodynamic findings in IHSS depend on the degree of functional obstruction produced by the septal hypertrophy.[8] Usually there is a subvalvular pressure gradient which may be accentuated by beta-adrenergic stimulation. The cardiac output may be increased, normal or decreased, depending on the severity of the obstruction; the left ventricular end diastolic pressure is typically elevated. Fifteen per cent also have some degree of right ventricular outflow obstruction, and a few cases of isolated subpulmonic stenosis have been reported.[3, 6] The typical angiographic findings are illustrated by Case Two *(Figure 2)*.

Although various series describe the common findings, the marked variability of familial IHSS is suggested by the two cases presented. In case one, the initial clinical findings of a holosystolic murmur

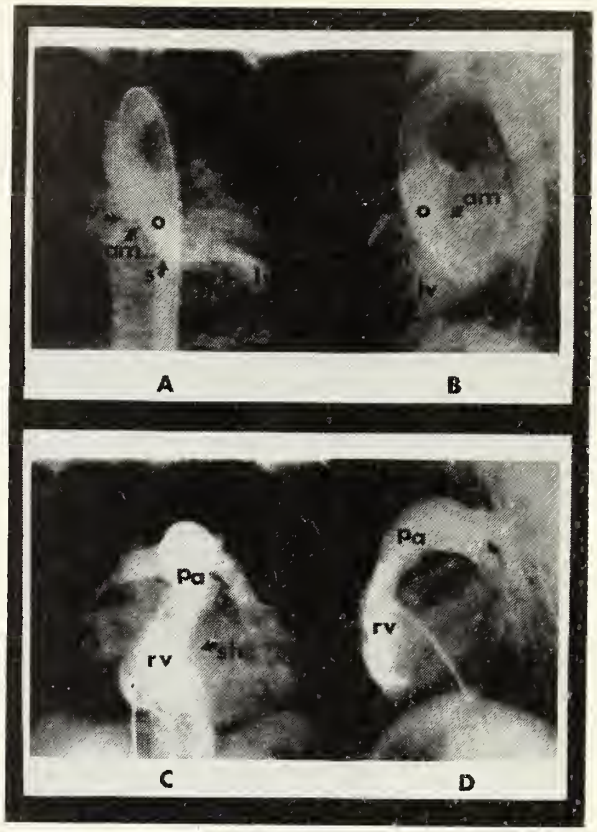

Figure 2. Patient Two. Biplane left ventricular angiogram (a) PA view, (b) lateral view. Biplane right ventricular angiogram, (c) PA view, (d) lateral view. av = aortic valve, am = arterial mitral valve leaflet, s = septum, sh = septal hypertrophy, lv = left ventricle apex, o = subaortic chamber, pa = main pulmonary artery, rv = right ventricle body.

along the left lower sternal border, an increased pulmonic second sound, and the EKG finding of right axis deviation and biventricular hypertrophy were consistent with a diagnosis of ventricular septal defect with secondary pulmonary hypertension. Since the child was asymptomatic it might have been inferred that this was not a hemodynamically significant lesion, and a decision simply to follow the child clinically might have been made. However, the presence of abnormal Q waves on the electrocardiogram was the clue that suggested IHSS *(Figure 1)*. In subsequent examinations with progression of the disease, the findings of a systolic ejection murmur at the right upper sternal border could have been confused with pulmonic valve stenosis or aortic valve stenosis. At cardiac catheterization, withdrawal pressure recordings from the main pulmonary artery to the right ventricle demonstrated a small infundibular systolic pressure gradient *(Table 1)*. Although there initially was no subvalvular pressure gradient across the left ventricular outflow tract, the left ventricular end diastolic pressure was abnormally elevated *(Table*

1). We now routinely use Lehman "end-hole" catheters in such cases, since a percutaneous multiple hole catheter will frequently miss such subaortic obstructions. The aortic pressure tracing also showed a bifid (double hump) contour. These findings suggested idiopathic hypertrophic subaortic stenosis with functional obstruction primarily of the right ventricular outflow tract by septal encroachment (reverse Bernheim effect). Septal enlargement was shown angiographically with apex obliteration of the left ventricle, and encroachment on the right ventricular outflow tract at an earlier age in the first case.

Case Two also presents certain findings which could cause confusion in making the diagnosis. This patient had clinical findings which were suggestive of both aortic and mitral valve involvement. Although there was no previous history of rheumatic fever, a holosystolic murmur along the left lower sternal border, a diastolic rumble at the apex, and an EKG showing left axis deviation and left ventricular hypertrophy might suggest the possibilities of rheumatic heart disease with mitral insufficiency or a ventricular septal defect. At cardiac catheterization, the patient had a small subaortic gradient across the left ventricular outflow tract, abnormally elevated left ventricular end diastolic pressure, and a bifid aortic pressure contour was shown *(Table 1)*. Angiocardiography showed septal hypertrophy into the left ventricular outflow tract,[14] with a mild degree of mitral valve regurgitation. This patient had idiopathic hypertrophic subaortic stenosis with functional secondary mitral valve insufficiency. The most recent cardiac catheterization shows angiographic and pressure changes in the right ventricle similar to Case One *(Figure 2)*.

The rationale for medical treatment of IHSS is based on the theory that any stimuli which increases cardiac contractility will also increase functional obstruction. Thus, obstruction is increased following such drugs as digitalis or isoproterenol, during or after muscular exercise, or following a premature ventricular contraction. Similarly, situations that reduce left ventricular systolic volume such as tachycardia, assuming the erect posture, Valsalva maneuver or nitroglycerin also increase obstruction. For these reasons, the beta-adrenergic blocking agents have been advocated for patients with IHSS. Propranolol has been shown to decrease obstruction in the basal state during exercise and increase exercise tolerance in symptomatic patients.[15] Propranolol has the additional benefit of decreasing the chances of tachycardia and lowering myocardial oxygen requirements.[16] Because of a relatively fixed cardiac output, patients with IHSS may have severe syncopal episodes or even die if tachycardia occurs (Patient One). The surgical treatment of IHSS would seem to be justified after a medical trial with beta-adrenergic blocking agents had failed to change the status of the severely symptomatic patient or documented cardiac deterioration is evident.

It is evident from the literature and from the two cases presented that the entity of familial idiopathic hypertrophic subaortic stenosis may present a variable clinical picture. In the pediatric age group the disease can easily be confused with cardiac abnormalities such as ventricular septal defect, pulmonary valve stenosis, aortic valve stenosis, and rheumatic heart disease. Since the child is initially typically asymptomatic, a tendency to procrastinate regarding definitive studies may occur. Although the incidence of early sudden death in familial IHSS has been recently found to be lower than previously reported, such a possibility exists.[3, 18] The ultimate long-term prognosis as well as optimal therapy await further experience in the pediatric patient. A recent case of an infant with this diagnosis on long-term Propranolol would seem to support the role of Propranolol as the drug of choice,[19] although surgical management also has strong support.[17]

Acknowledgment

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(Continued on page 352)

Medical HISTORY

Joseph Brenneman, 1872-1944

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"It is too obvious to require repetition that medical knowledge is not the product of one age or even one century. We have unconsciously assimilated medical theories and facts which have come to us through the centuries. Of course, the science and practice of medicine is not static, the discovery of new facts and procedures constantly increases our medical horizon and our usefulness. We are not unmindful of the fact that the healing art has passed through stages of empiricism and darkness; nevertheless, in all periods there arose masters who made contributions which have lasted throughout the ages. To these heroes we owe gratitude and pay homage."—Isaac A. Abt, 1942, from the foreword to *Pioneers of Pediatrics* by Abraham Levinson.

JOSEPH BRENNEMANN was born near Peru, Illinois, on September 25, 1872, and received his preliminary education at a district school (later named the Brennemann School in his honor) across the road from his country home. While working on a farm, he prepared himself for college and entered "by a close shave and only by the grace of my knowledge of German."

His training began at the University of Michigan, where he did his premedical work and received the PhB degree in 1895. He taught for one year, and then attended the Northwestern University Medical School in Chicago, Illinois, graduating in the class of 1900. His course was interrupted by the illness of his brother. He went to be with him in Texas and, while there, taught in a Mexican district school. This was a one-room school in an adobe house where he taught his pupils some English, and they taught him some Spanish. His third year in medicine was spent at the Gross Medical College in Denver (five months' course). A two-year internship at St. Luke's Hospital in Chicago followed, and then he began his general practice on Chicago's south side.

In 1903, he became associated with Dr. Francis X. Walls, then chairman of pediatrics at Northwestern, as instructor in clinical pediatrics. That was an important year, for in 1903, Dr. Brennemann married Bessie Darling Daniels. There were three children: Mary, Barbara, and Deborah; and, at his death, seven grandchildren.

After some years of practice, he decided to become a pediatrician. So in 1910, Brennemann left for Europe, where he studied for three months in Germany and Austria. He returned to Chicago and continued his work at Northwestern in the outpatient department as attending physician, and at the St. Luke's Hospital and Wesley Memorial Hospital. His interests were in feeding problems in infants, and he contributed to the literature in this area. In fact, it was his paper published in 1911, "Contributions to Our Knowledge of the Etiology and Nature of Hard Curds in Infants' Stools," which settled a controversy that had been going on for years. Brennemann always insisted that this paper was his best piece of work, despite wide recognition he received for his many other papers. The experimental method used for his paper on "curds" is very interesting. Brennemann was "fortunate" enough to have an experimental subject in the person of his sister. She had the ability to regurgitate at will; thus, she was used to taking milk and regurgitating it at certain times for analysis.

Brennemann went about his work at Northwestern with devotion and thoroughness. For instance, in 1915, when he was the attending physician at the dispensary, he spent two hours a day, six days a week working there. In addition, he taught a section of junior pediatrics and made pediatric rounds daily at Wesley and St. Luke's. One might consider this not a heavy teaching load, but he was giving all this time without financial remuneration and was dependent upon his private practice for financial support.

During these years, Brennemann was secretary of the Chicago Pediatric Society for six years and its

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The opinions contained herein are those of the author and not of the U. S. Army Medical Corps.

Joseph Brennemann

president for one. In 1921, he left his position at Northwestern to be chief of staff at the Children's Memorial Hospital. The story of his acceptance of this position is an intriguing one.

Dr. Frank Billings made an appointment to see Brennemann at the latter's office. Brennemann was not informed of the nature of Billings' visit but was convinced of its importance just because of the fact that the "great Billings" was coming. The office attendant was notified to let Billings in the moment he arrived, and one of the examining rooms was kept free of patients so there would be no delay. Brennemann, a calm man, was nervous indeed. Billings explained that he had come to offer Brennemann the position of chief at Children's and professor of pediatrics at the University of Chicago. Brennemann was overwhelmed and could not give an answer to the offer. He considered himself unfitted for the job and, in fact, could only give his notice of acceptance after several weeks of deliberation. Once this decision was made, the apprehension did not subside. In 1921, he began his work as "Chief," but on the first day stayed only about half an hour because he felt unequal to the task. The next day, he managed to stay somewhat longer. Only several weeks' time allowed him to really feel at home in the job and with the work. Part of his fear arose because he felt he knew nothing of children's hospitals, his only experience being with small pediatric wards of general hospitals.

This stumbling block overcome, Brennemann at once began writing and in a short time was nationally known. His position at Children's Memorial Hospital according to Reichert, was "one of the most important things that has ever happened to the children of America—probably to all children, since it gave him an opportunity to study large numbers of children, and it supplied him with a forum from which he could demonstrate his great talents and share his knowledge with the pediatric world." From 1921 to 1930, Brennemann spent about three hours each day at the hospital without any financial remuneration. In 1930, he became the first full-time staff member. He was chief of staff until 1940, when he retired and went to the Children's Hospital of Los Angeles where he was chief of staff and professor and head of the Department of Pediatrics at the University of Southern California Medical School. He retired from here in 1943, but did not give up his work in pediatrics. He spent a great deal of time editing his *Practice of Pediatrics*. In April 1944, he served as visiting physician at Milwaukee Children's Hospital, and in June he read a paper at the American Medical Association Section of Pediatrics meeting.

It was at his home near Reading, Vermont, that on July 2, 1944, Brennemann died with a myocardial infarction. This illness was brief. He had complained of slight pain in his chest and left arm for a few days, and about 12 hours before his death summoned his physician. An electrocardiogram was taken, and the doctor returned with the tracings. Brennemann put on his glasses to evaluate them when he suffered the fatal attack.

What did Brennemann accomplish as a pediatrician?

He was recipient of all the honors of a pediatrician. He was a member of the Chicago Pediatric Society (president, 1918), American Academy of Pediatrics, and American Pediatric Society. He was admitted to the latter in 1918, was recorder and editor in 1921-22, and its president in 1930. In addition, he was honorary member of the Society for Pediatric Research, and a corresponding member of the British Pediatric Society.

Brennemann's greatest achievement was the editorship of and his contributions to his *Practice of Pediatrics*. In this position, he prepared the most monumental work of pediatrics of his day. The amount of work he put into this endeavor was massive. He informed each contributor (125 of them) as to the amount of space which each would have available. However, the articles submitted were three to four times the specified length, other physicians were late in sending in their essays, some were poorly written. However, Brennemann did not give up easily; he felt

personally responsible for the quality of the articles submitted, so he revised all and even rewrote some of them.

When the books appeared, they were received with enthusiasm and praise, and they won Brennemann great fame. These volumes were continued after his death under the editorship of Irvine McQuarrie, and currently under Vincent C. Kelley. Their publication was indeed a milestone in medical history, and they are still regarded as the "Bible of Pediatrics."

In 1919, appeared a book by Brennemann, *Infant Feeding: Maternal and Artificial.* This was a thorough work describing the type foods to feed a baby and the methods by which it should be done, and describing feeding and nutritional disturbances. This could be purchased by the public for practical use.

Stanley Gibson describes Brennemann as a teacher:

His fame as a teacher equaled or exceeded that which he enjoyed as a writer. He spoke clearly in good voice, and was always completely honest to the minutest detail in the presentation of any clinical case. He had a superb gift of showmanship. He knew how to set contrasting cases off against one another; he knew how to highlight the essential features which made the case worthwhile; and he knew the value of suspense in holding the interest of his audience.

Some of his more outstanding papers were on the following topics. As previously mentioned, he was, for some years, keenly interested in the nutrition of children. Interest in this area hit the country somewhat later, and the child became overlooked in the new fad of measurements—height and weight. This led Brennemann to write his paper on the "Psychological Aspects of Nutrition in Childhood."

He wrote:

Parents have been led to think that their children must be standardized, that they must conform in weight and height to an assumed norm as determined by averages expressed in tables of weights and measures that are now found in all baby books, most nurseries, and on the walls of the offices of nearly all physicians who care for children, except for mine. A few pounds above, even more a few pounds below the figures, call for standardization. The mother's eyes are fixed on the scales as the final arbiter of nutrition, color, tissue turgor, activity, endurance, race, individual and family body architecture are lost sight of as the real measures of nutrition.

In another similar "philosophical article," he presented a message that is well worth considering today. Our civilization progresses by one mass purge of the old and overindulgence in the new. Instead of stepping lightly into a new concept and receiving its benefits from proper use, we drown ourselves in the new ideas. All at once, psychology was discovered in the child, and everyone attached the new words to their every thought. Brennemann's paper was entitled, "The Menace of Psychiatry." He writes in his summary:

What I have attempted to point out is the menace that lies, inescapable psychologically in the too intensive injection into the lay mind, which cannot usefully assimilate it all, of a mass of complicated, confused, and as yet unsatisfactorily organized material in a domain that requires "wary walking" if more harm than good is not to be done. I am a little afraid that in our efforts to meet a problem, we may be making more problems to solve. I might add that the pediatrician has probably done this more often than anyone else. It is a danger inherent in all medicine.

These thoughts met with worldwide approval and, as a result, Brennemann was invited to London to give a paper before the Section on Psychiatric Medicine of the Royal Society of Medicine.

Children's Memorial Hospital grew and prospered under Brennemann's leadership. He was not happy with the affiliation for teaching at Children's Memorial. At this time, the hospital was used by the University of Chicago. He wrote in the 1931 annual report:

It has always been a matter of regret that the facilities of the Hospital could not be used more extensively for teaching purposes. It is a well known fact that teaching and efficiency go hand in hand. Several monthly courses a year for graduate physicians, and our affiliation with The University of Chicago have not and probably never will satisfactorily fill this need. For this reason, arrangements have been made for regular attendance of senior medical students, the most desirable teaching material, from the nearby Northwestern University Medical School. It is a reciprocal service equally appreciated, I believe for both parties. Without entering into any affiliation with Northwestern University, and without in any way disturbing our affiliation with the University of Chicago, we have now two amphitheatre clinics a week of 1½ hours each for ten men daily for two hours in the out patients department medical clinic, a somewhat smaller group once a week in the out patients department orthopedic clinic, and two clinical clerks in the wards. As a courtesy, and as a matter of reciprocal service, we have asked Dr. Abt, the head of the Department of Pediatrics of Northwestern University Medical School to conduct one of the amphitheatre clinics.

Thus began the affiliation with Northwestern University, which grew into the massive use seen today by Northwestern.

Brennemann constantly advocated improvement in the quality of care given at and in the facilities of the Children's Memorial Hospital. He was, even in 1931, eager to form a fellowship program.

I had intended to speak—of the great good that could be accomplished some time in the future by the establishing of fellowships, or part time positions to attract

good men to certain departments, men who have spent all of their money on brains that find no eager world on which to use them with sufficient immediate profit to make a living.

As a person, Brennemann had many interesting traits. One was a lack of self-confidence, as he put it, "a congenital inferiority complex." He felt that the men whom he had asked to write for his *Practice of Pediatrics* would not write for him. His feelings concerning his position as chief of staff have already been noted.

This was a small price to pay for his kindness, sincerity, and other traits. Gibson wrote:

Over and above Dr. Brennemann's brilliant intellectual achievements was his solidarity of character. One never doubted his sincerity. He could not tolerate pretense or sham. He stood for what he believed to be right and had the courage to voice his convictions. He cared not at all for social life in the usual sense, but he was supremely happy in a small gathering of congenial friends. His friendship knew no bounds. He was no less the friend of the errand boy at the hospital or of the humblest dispensary patient than he was of his medical associates.

Yet it was the young men and women in medicine who were nearest to his heart. He was never the "big professor." He worked with his residents and always insisted that he learned more from them than they from him. His attitude is exemplified in the dedication of his *Practice of Pediatrics:* "In a very affectionate sense to that keen, earnest and loyal group of young men and women whom I have tried to teach and who have taught me so much, my interns and residents of many years."

Dr. Gibson describes him further:

It is difficult to know what qualities make for greatness in the field of medicine. That Dr. Brennemann had certain qualities which set him apart from the majority of his colleagues all who knew him well will agree. He had a rare independence of mind. The new in medicine had to be subjected to the severest scrutiny not once but many times before he was willing to accept it. He never followed the crowd. In fact, he sought on numerous occasions to turn his colleagues from accepting too easily the current fashion in pediatric thought.

Brennemann had a consuming interest in many areas not medical. About two years before his death Gibson, in a friendly meeting, asked Brennemann if he had read a certain article in the *Reader's Digest.* The answer Gibson received was, "No, I do not believe I have come to it yet. I have the magazine with me in the Spanish edition and I am a bit slow in reading it. I don't know why the devil a man of 70 should be trying to perfect his Spanish but anyway it's good fun."

Then Brennemann quoted a phrase in Latin which meant "nothing of human interest is foreign to me." Even though this Latin phrase was quoted by him on several occasions and half-jokingly, it was a characterization of Brennemann. When ill once, he spent his time reading a large text in geology; his reading interests were indeed varied—from Homer in the original to the philosophy of Kant in German.

Gibson recalls an example of Brennemann's humor:

One morning when I walked into his office he handed me a clipping from his home town paper. The editors had learned somewhat belatedly that their native son had made good. They related his numerous achievements and honors. In conclusion they stated that his reputation was such that he was now regarded as the second greatest pediatrician in the world. I handed the clipping back to him and he glanced at me slyly, waiting for my comment. The situation struck me as highly amusing, and I was determined that he should speak first. After a long silence he finally said, "Who in hell do you suppose that other guy is?"

The American Pediatric Society described Brennemann as a "straightforward man of good old German stock, singularly devoted to his work, one whose life is characterized by simplicity of manner and the greatest integrity. He is a clinician of the highest order and possesses great administrative ability. He is a high minded man, capable of loyal friendship, and loved by his colleagues and students."

Today, Brennemann's portrait hangs in the outpatient department of the Children's Memorial Hospital, and the Joseph P. Brennemann Library has a fine collection of books on pediatrics and related areas. The Children's Memorial Hospital Library was named after this famous pediatrician in December of 1940.

Acknowledgments

The photograph is reproduced with permission of the Children's Memorial Hospital.

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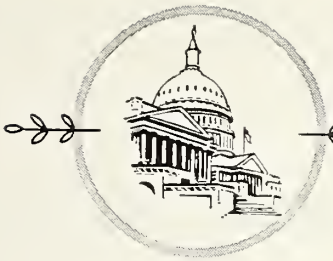

Socio- ECONOMICS

Disability Insurance Under Social Security

DWIGHT LAWSON, M.D.,* ***Topeka***

WHENEVER A PHYSICIAN is asked to furnish a medical report in connection with a patient's claim for Social Security disability benefits, it is a reminder that Social Security is not just for the retired—it also provides important financial help for people who cannot work because of a serious illness or injury. Currently, over 3 million men, women, and children receive Social Security disability checks every month because someone in the family—usually the breadwinner—is disabled. Their payments total almost $5 billion a year. In addition, more than 76 million working men and women are insured for disability benefits as a result of their earnings—wages or self-employment—under Social Security. In Kansas, these benefits are of significant economic importance and, as of December 1972, there were 14,323 workers with 10,426 dependents on the rolls. Their payments amounted to $2,558,000 monthly; since then, this amount was increased by 20 per cent, currently exceeding $3,000,000 monthly in payments to these disabled workers and their dependents in Kansas. Beginning July 1, 1973, full Medicare protection will be extended to persons under age 65, who for at least 24 consecutive months have been receiving monthly Social Security benefits because they were disabled.

A person under 65 can receive monthly disability benefits if he has a physical or mental impairment severe enough to prevent him from doing any substantial, gainful work for a year or longer. Benefit amounts based upon a person's earnings under Social Security range from $84.50 to $345.50 a month for the disabled worker alone, and the maximum monthly benefit for a disabled worker with a family is $620.40.

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From a Small Beginning

The original Social Security Act of 1935 provided benefits only for the retired worker. It was not until 1954, when the disability "freeze" provision was added, that the law gave some protection to the disabled worker. Under the freeze, years when a worker earned little or nothing because of disability were not counted against him later in deciding upon his eligibility for retirement benefits, or in figuring his retirement benefit amount. To be eligible for the freeze, the worker had to have a disability that was expected to be of "long-continued and indefinite" duration.

Two years later, monthly cash benefits were provided for disabled workers aged 50 to 64, and also for the disabled adult sons and daughters of retired or deceased workers if the son or daughter had been continuously disabled since childhood.

Over the years, the program has been further improved. The minimum age limit of 50, for payment of benefits to disabled workers, was eliminated; "long-continued and indefinite" duration was changed, so that an insured worker could be eligible if his disability had lasted or could be expected to last for at least 12 months; fewer years of covered employment were required for a young worker to be insured for disability; and benefits were provided for disabled widows (between ages 50 and 60) of covered wage earners. The latest change is, of course, Medicare protection for disabled persons under 65.

Who Can Get Benefits

Social Security disability benefits can now be paid to the following:

A disabled worker under 65 and his family, if

he has worked under Social Security for a certain length of time, ordinarily 5 to 10 years preceding the onset of disability. (Special provisions apply to workers disabled by blindness allowing them to qualify with even less work under the program.) For the worker who becomes disabled before he reaches 31, the work requirement ranges down with age to as little as 1½ years.

A person continuously disabled since childhood (before age 22), if one of his parents (in some cases a grandparent) who is covered under Social Security retires, becomes disabled, or dies. The mother of the disabled son or daughter may also receive monthly benefits as long as she has the child in her care.

A disabled widow 50 or over, if her late husband was covered under Social Security, and if she meets the specified level of medical severity. This also applies to disabled dependent widowers and certain disabled surviving divorced wives.

Reporting Medical Evidence

When a patient applies for benefits, he is asked to submit medical evidence to support his claim. The evidence usually consists of data from the records of his treating physician, clinic, or other medical source. Our experience with the disability program in Kansas indicates that in about three out of five cases no further medical development is needed, because the treating source already has enough information on record to provide a good picture of the applicant's condition and how it limits his ability to work.

This information may be requested on the patient's behalf by a Social Security office or, more often, by the Disability Determinations Unit. This is the full name of the agency in Kansas that evaluates Social Security disability claims for Kansas residents. Like other state agencies throughout the country which work with Social Security in the disability insurance program, the Disability Determinations Unit in Kansas includes both physicians and trained disability examiners on its professional staff. They form a balanced team of medical and non-medical people who can handle anything from a strictly medical issue to a complete assessment of the vocational factors which bear on the disability decision.

With the assistance of the staff of reviewing physicians, the Disability Determinations Unit endeavors to make these requests for medical information relate as directly as possible to the condition which the claimant states is the cause of his disability. The goal of the individually tailored request is to ease the medical reporting burden of the busy physician or clinic, without jeopardizing the claimant's right to have his case decided on the basis of all relevant information available.

The evaluating physician in the Disability Determinations. Unit never sees the patient. He depends heavily on information supplied by the physician or clinic to assess the severity of the applicant's impairment, its expected duration, and the extent of his residual functional capacity. The disability decision, therefore, rests largely on the quality of the medical evidence obtained. A detailed report from the treating source, including objective findings and laboratory procedures, will usually be sufficient to evaluate the claim and make a decision.

For example, if the patient experienced a myocardial infarction, this agency would look to the report submitted by the treating source for such information as date of occurrence, place and duration of the hospitalization, as well as results of x-rays, electrocardiograms, and other laboratory studies. Serial EKG tracings should, whenever possible, accompany the report, so that our staff of physicians may also have the benefit of reviewing this essential documentation. Equally important is the medical history, including onset of chest discomfort, relationship to effort, intensity, location, radiation, regularity, and to what extent relief is obtained by rest or medication. If a report does not contain all the findings necessary to make a proper decision, one of the reviewing physicians may re-contact the medical source. However, the additional time required may delay the patient's claim and can add up to a significant additional program expense.

The physician can help speed the decision on his patient's claim by reporting all relevant data about the patient's medical condition as promptly as possible.

Establishing the onset date of disability—often a key factor in determining the beginning date and amount of the claimant's benefits—is frequently difficult. Therefore, it is extremely helpful if the reporting physician includes the date of each important fact or finding. To save time, he may enclose photocopies of pertinent sections of the patient's chart, or of hospital or consultant's reports.

Criteria for Evaluating Disability

In making disability determinations, the agency uses medical criteria developed by the Social Security Administration to insure uniform evaluation of all applicants no matter where they live, and to help simplify and speed the decision process. These criteria were worked out with the aid of practicing physicians, major medical organizations, and SSA Medical Advisory Committee.

Generally, a claimant who is not working can meet the Social Security definition of disability if he has an impairment or combination of impairments which are the same as, or medically equivalent to, any

(Continued on page 354)

The President's Message

The decision on the Professional Standards Review Organization (PSRO) has already been made. The alternatives to doing peer review under the Professional Standards Review Organization are not exactly clear at this point, although the law states that this may be done by medical doctors if they can be structured in the right way. The professional standards review law will materially affect your practice, your relationships with your patients, and your income. Your practice will be standardized under this system and you will be subject to norms as determined by people other than yourself.

Physician profiles will be developed as they have been done in the past, and you will have very little access as to what this profile says. The Professional Standards Review Organization will completely alter the present structure of our medical societies. If you were a social planner, and were attempting to restructure the health care delivery system, one of the first things that you would do, would be to completely break down the medical associations as they are presently structured. Although most medical societies are very loosely organized, they still function as a single unit, and this structure must be destroyed if you, as a social planner, are going to succeed in implementing your plan.

It is extremely important that the physicians individually and collectively in Kansas understand what the Professional Standards Review Organization is, what it does, how it operates, and who controls it.

Thomas F. Taylor

President

The Truth Shall Make Your Fee

Being of a disorderly and pedestrian mind, we have adopted the habit of attempting to reduce some of the perplexities of life to a root cause which will permit us to resolve all of the associated problems with one simple basic adjustment. The numerous griefs besetting the medical profession are a case in point and we think we can see a common element in the dysrhythmias which afflict the profession. It is simply this: we have yet to produce a totally satisfactory method of equating medical service with material compensation. The forms of medical payment have been numerous: the coin of the realm, other services, a chicken or an upside-down cake, heartfelt thanks—or nothing. The offering and acceptance of these payments, however, have not brought us to a stable, dependable system, and virtually every conflict in the medical world can be traced back to our inability to establish a definite relationship between the medical function and monetary exchange.

This is due not so much to the fluctuation and instability of the various media of exchange, or the unpredictable market for services, as to the fact that we have never been able to settle on the worth of life and its handmaiden, health.

Physicians themselves have no clear understanding of how to set their fees—theories, systems, plans—yes, but not understanding. In spite of countless physician man-hours spent in devising relative value schedules, there is unspoken awareness that these are not solving the problem but simply providing a delay against the incursion of outsiders into the fee establishment. The physician knows too well the difficulty of reconciling his expenditure of physical and intellectual energy with the consequent benefit to the patient. A brief office visit and minimum laboratory data may serve to establish a diagnosis which saves a patient's life. Or an hours-long, complicated surgical procedure requiring an expert staff and expensive, sophisticated equipment may result in sustaining the patient, not necessarily comfortably, for a few more units of time.

The internist who makes a diagnosis resents the modesty of his return as compared with that of the surgeon who corrects the defect. The generalist resents that the breadth and depth of his care commands a smaller fee than that of the specialist whose service is transient and patient-contact shallow. The physician feels justified in accepting a given fee for a relatively small service knowing that he will often have to accept the same fee for a much greater service. He rankles under the knowledge that his capacity to attract patients—and their fees—is less than that of his more personable colleague of inferior ability but superior rapport.

As life has been extended, the emphasis has changed. A century ago, the seriously ill or injured patient was expected to die and his survival could be counted a miracle. Today, with miracles commonplace, his survival is expected and death is counted a failure, suspicious of inadequate service. The physician is expected to guarantee life and health—and for a predetermined fee. The courts offer a seemingly unilateral interpretation. In judgments on the death of a mother, her value has been determined by the estimate of the cost to the husband and father for the replacement of her services. An injured patient is compensated in proportion to his loss of productivity blamed on the physician. A damaged infant receives compensation based on the cost of his maintenance for an estimated life span. But can the physician who saves the life of a mother charge on the basis of her continuing value to the family? Can the physician who returns an ill or injured person to productivity expect a continuing share in that productivity? Can the physician who accomplishes the delivery of a healthy baby from a high risk pregnancy charge in proportion to the subsequent value of that child? At least, this suggests that the physician's successful service is measured on a different scale from his failures (if they are that), and thus reflects the problem of placing monetary equivalents on life and health and the physician's function.

The argument continues as to whether we have a shortage of physicians or just a maldistribution. The

latter is certain and the former is made true, in some areas at least, by this fact. A shortage is certainly operative in the fee establishment since it affects the physician's time and availability and the relative value of his service (as in any shortage market). But if it is a matter of inequitable distribution, it still relates to the fee problem since the deprived areas evidently cannot provide the inducement in compensation offered by the supplied areas.

This preoccupation with compensation would be interpreted by those antagonistic to the profession—or its system—as evidence of avarice. (Not so. Compensation is ultimately a biologic necessity if the organism is to survive. The complexity of our society is simply the complexity of the compensatory systems that have evolved.) But the appearance of avarice on the part of the physician is fostered by the failure of the patient to consider the service worth the price. This, more often than not, relates to failure of communication between physician and patient. Conversely, if the physician has maintained good communication, the fee will be understood and accepted even if the service is less than successful.

Efforts to establish a truly equitable system of medical charges have always been contentious. The physician approaches it as a qualitative problem with quantity secondary (but present). Health care plans have approached it as a quantitative problem with quality secondary (but present). The best the physician has come up with is the fee-for-service idea which is now under siege by those who refuse to accept a plan in which the physician seems to be the sole arbiter. The physician must defend a plan with many inherent uncertainties and variables. How does he arrive at the evaluation of his service? From a mash of ability, responsibility, time, availability, overhead, and (the most difficult of all) his estimate of the value to the patient, he tries to distill a palatable solution.

And how does the patient interpret the value of the service? His ability to pay? Some relative value of the identity of the patient—the father, mother, or child? The speed and comfort with which the cure is effected? The return to previous or better health with no risk of recurrence? The emotional support supplied along with the specific therapy?

It is evident that, even before we get to the question of compensation, the healing effort is a unique entity. It is at once physical, spiritual, emotional—and an economic force. For all our efforts to do so, we cannot really measure it, weigh it, package it. It refuses to be consistent and controlled as long as we have no measure of the value of the ultimate objective.

This posing of questions is an easy type of treatise to construct but obviously gives no immediate answers. In his efforts to lead his reader through a mental maze, the writer cultivates an attitude of profundity to obscure his inability to solve the problem he has proposed. If our thesis is correct, we can offer only the prospect that the problems will be with us until physician and patient are given a mutual understanding of the value of life—and one wonders if it is of this world.—*D.E.G.*

LIBRARY SERVICES AVAILABLE TO PHYSICIANS

Clendening Medical Library at the University of Kansas Medical Center will continue to provide the types of services initiated by the Kansas Regional Medical Program before federal funding for its Library Systems Project was discontinued June 30, 1973. Minimal charges will be necessary, and copies of the fee schedule for services have been mailed to Kansas physicians and other health care professionals known to be interested in the services. Some changes in charges may be made at a later date after evaluation by the State Finance Council.

The following services are currently available:

Quick reference questions—no charge.

Loan of books or other materials—no charge.

Photocopy of journal articles—no charge except for recent articles in readily accessible journals.

Searches of the literature on topics of concern—$2 charge per search.

A toll-free telephone line will be installed and announced as soon as possible. Requesters may inquire as to its availability by dialing Directory Assistance for Inward WATS at the following number: 1-800-555-1212. The line will be listed under the name of Clendening Medical Library State Services. Requesters in the Kansas City area not associated with the University of Kansas Medical Center may call the following number: 831-7313. Requests may also be sent by mail to the following address: Clendening Medical Library State Services, University of Kansas Medical Center, Kansas City, Kansas 66103.

KMS Obituaries

MICHAEL J. COX, M.D.

Dr. Michael J. Cox, of Dodge City, died July 3, 1973, at the age of 73. He was born January 22, 1901, in Worcester, Massachusetts.

Dr. Cox was graduated from the Tufts University Medical School, Boston, in 1927. He had practiced in Dodge City since 1950.

Surviving Dr. Cox is his wife.

BERTRAND I. KREHBIEL, M.D.

Dr. Bertrand I. Krehbiel, 70, of Topeka, died July 20, 1973. He was born September 15, 1902, in Newton.

Dr. Krehbiel was graduated from the University of Kansas School of Medicine in 1927. He practiced in Topeka since 1930.

Survivors include his wife and two daughters.

LAWRENCE A. PROCTOR, M.D.

Dr. Lawrence A. Proctor, 68, of Parsons, died June 22, 1973. He was born February 3, 1905, in Parsons.

Dr. Proctor was graduated from the University of Kansas School of Medicine in 1933. He began his medical practice in Parsons in 1934.

Surviving Dr. Proctor are his wife, a daughter, and a son.

AMA House of Delegates

Summary of Actions Taken at the 122nd Annual Convention, New York, New York, June 24-28, 1973

Resolutions requested by the Kansas House of Delegates were all covered by other resolutions, and were acted upon. The President and the President-elect made speeches which were well received, but since those are covered in the *American Medical News,* they will not be repeated here. The AMA House of Delegates—confronted with the largest business agenda in the Association's history—acted on a wide range of issues during the 122nd Annual Convention, which affect physicians in their relationships with government, medical schools and hospitals, and with the public. The issues ranged from PSRO's and wage-price controls to institutional licensure and the need for more primary care physicians. Meeting for a total of 18 hours and 51 minutes, the House acted on 84 reports and 179 resolutions, for a total of 263 items of business.

Delegates selected Malcom C. Todd, of Long Beach, California as President-Elect. Also elected were E. Bryce Robinson, Jr., Alabama, Vice President; Tom E. Nesbitt, Tennessee, Speaker of the House; William Y. Rial, Pennsylvania, Vice Speaker of the House.

The Kansas recommendation that every delegate be placed on a council of the AMA was referred to the Council on Long Range Planning. The House acted to request an end to the doctor draft. It was also voted to consider a new definition of the term "provider."

Three resolutions were introduced declaring the AMA should consider its membership in the World Medical Association. These resolutions were not adopted and by this failure to act, the AMA has withdrawn from the World Medical Association.

The request for a standard health insurance form was referred to the Council on Medical Service. The Board of Trustees were requested to study the question of paying AMA dues directly to the AMA.

Food and Drug Regulations

Resolved, That the American Medical Association continue to protest those proposed and current regulatory activities of the Food and Drug Administration which have the effect of restricting the use of prescription drugs to the condition and dosage recommendations stated on the "official labeling" and printed package insert or which threaten to interfere with the exercise of a physician's professional prerogatives in selecting the drug of choice for a patient; and be it further

Resolved, That the American Medical Association study the possibility of proposing modifications in the Food, Drug, and Cosmetic Act to correct current problems; and be it further

Resolved, That the American Medical Association recommend to the Food and Drug Administration that practicing physicians representing medical specialty societies be members of the committee of the FDA to make final decisions as to the usefulness and effectiveness of the drugs; and be it further

Resolved, That in continuing to work closely with the Food and Drug Administration in the fulfillment of its statutory responsibilities, that efforts be made to develop an effective system of communicating to and considering the views of practicing physicians and medical specialty societies when action is proposed that may result in removal of frequently prescribed drugs from the market.

PSRO and Peer Review

Resolved, That the Secretary of Health, Education, and Welfare be informed that the only organization which can give qualified peer review for physician services to the patient, physician, government, and taxpayer are those composed of practicing physicians, whether these are state or local groups; and be it further

Resolved, That since many of these practicing physician groups are functioning successfully, with multiple approaches, as peer review organizations, the regulations be so written to authorize these existing peer groups to continue their review as PSROs or as functioning units of PSROs, thus partially alleviating the unnecessary and costly implementation of new agencies as PSROs; and be it further

Resolved, That although it is recognized that repeal or modification of PSRO legislation ultimately may be required to preserve the high quality of patient care, the American Medical Association should oppose any facets of this current legislation which act to the deterioration of quality care, publicize such deleterious facets, and place highest priority on developing and pursuing appropriate amendments to preserve the high quality of patient care.

Certificate of Need

Resolved, That the American Medical Association reaffirm its continuing support of voluntary planning that preserves decision making at the local level; and be it further

Resolved, That if state certificate of need legislation is enacted, it should contain the following provisions: (1) final authority rest within a board with representation by physicians in active medical practice; and (2) there be provision for adequate input by physicians in active community practice; and be it further

Resolved, That these actions be forwarded to the Secretary of Health, Education, and Welfare with the request that they be included in the regulations for implementation of the Comprehensive Health Planning Act.

QAP—Quality Assurance Program

Resolved, That the American Medical Association reaffirm its advocacy of peer review, including utilization and quality of care review, and of continuing medical education activities; and reaffirm further that these elements for the accountability of patient care are the responsibilities of the attending physician; and be it further

Resolved, That the American Medical Association express to the American Hospital Association its grave reservations about the Quality Assurance Program because of its potential for lay control of medical practice.

Physicians on Hospital Boards

Resolved, That where legally permissible, physicians actively practicing in the community who are voting members of the medical staff shall be eligible for, and should be accorded, membership on hospital governing bodies and their action committees, with full voting privileges, in the same manner as are other knowledgeable and effective individuals; other physicians also should be considered eligible for membership on governing bodies; and be it further

Resolved, That state and local medical societies be urged to seek removal of legal and other barriers to medical staff membership on hospital governing bodies wherever these exist; and be it further

Resolved, That physician representation on hospital governing bodies be significant in number and not merely token representation; and be it further

Resolved, That the Joint Commission on Accreditation of Hospitals be urged to ascertain from the JCAH inspectors the effectiveness of communication between governing bodies and medical staffs of hospitals as a requirement for accreditation; and be it further

Resolved, That the American Medical Association declares that any proposal or arrangement between a hospital board of trustees and its medical staff that conflicts with the AMA Principles of Medical Ethics is improper; and be it further

Resolved, That unilateral changes in medical staff bylaws by hospital boards of trustees are also improper; and be it further

Resolved, That the AMA suggest that the following preamble be included in all medical staff bylaws:

> The hospital and the medical staff have a duty to cooperate in their mutual responsibility of assuring the highest quality of patient care standards within the hospital. Only physicians can practice medicine under the laws of the state. In those areas in which medical judgment and the evaluation of professional competence are involved, the hospital has a duty to rely upon the judgments and recommendations of the medical staff, to cooperate and to provide needed assistance with full understanding that the primary responsibility is that of the medical staff.

Social Security Benefits

Resolution 166 requested the AMA to assist in correcting inequities in the present methods for calculating Social Security benefits for physicians. Such benefits are now calculated from the year 1950, even though physicians did not come under Social Security provisions until 1965. Hence, physicians are penalized by 15 years of "zero contribution" in the computation of benefits. This resolution was adopted and referred to the Board of Trustees for implementation.

Institutional Licensure

Report H of the Board of Trustees called for the American Medical Association to oppose the extension of institutional licensure in lieu of individual professional licensure to physicians and nurses. This report was adopted.

Family Medicine

There were several resolutions relating to family medicine. Most of these were referred to the Council on Medical Education. One resolution passed by the House recommends that medical schools encourage 50 per cent of medical students to enter primary care.

John C. Mitchell, M.D., Salina
George E. Burket, Jr., M.D.,
Kansas City, Kansas
AMA Delegates

Join
KaMPAC

Medical-Legal Page

Pregnancy After Tubal Ligation

In a malpractice action arising from an unsuccessful tubal ligation, the Deleware Supreme Court held that the trial court had improperly allowed the jury to decide whether or not public policy barred recovery of damages.

A woman who had five living children underwent a tubal ligation. The sterilization procedure was unsuccessful, and she became pregnant again.

The woman, her husband, and the five children brought action against the physician and hospital. They sought to recover damages for the wife's pain and suffering during the pregnancy, the cost of the sterilization procedure, the husband's loss of consortium, the deprivation to the children of the care and support they would have received in the absence of the sixth child, the medical expenses incurred in the pregnancy, and the cost of caring for the sixth child.

The physician and hospital moved to dismiss the complaint, contending that it was an action for "wrongful life," which was not recognized in the state. The trial court denied the motion.

On appeal, the physician and hospital contended that no compensable injury existed. They said that there were policy considerations prohibiting recovery and that even if there were an injury it was outweighed by the blessings of a child.

The supreme court found that the trial court had not decided the question of whether the action was barred by public policy. Although the court found that there was no policy resting on the desirability of procreation that barred recovery, it did not consider other possible objections. Instead, the trial court shifted the question to the jury.

The supreme court found that the judge could not shift his burden of decision to the jury and that the opinion and order of the trial court were a nullity because no substantial legal issues had been decided. Dismissing the appeal, the court said that since some of the rulings of damages in the opinion were probably too speculative, the best interests of justice required that the entire opinion be refused recognition as the law of the case.—*Wilmington Medical Center, Inc. v. Coleman,* 298 A.2d 320 (Del.Sup.Ct., Oct. 25, 1972).

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Editor's Note: A previous decision was reported in *The Citation,* Vol. 24, No. 4, p. 49.

Unsuccessful Vasectomy

In an action for malpractice arising from an unsuccessful vasectomy, the statute of limitations commenced to run on the date the patient discovered that the operation had failed or the date on which in the exercise of ordinary care and diligence he should have discovered it, the Texas Supreme Court ruled.

After his wife had given birth to two deformed children, the patient consulted a doctor of osteopathy for the purpose of having a vasectomy. The osteopath performed the operation in July, 1966. A month after the operation, the osteopath assured the patient that he was sterile. In September, 1967, the wife discovered that she was pregnant. In January, 1968, she gave birth to another deformed child, who died nine months later.

In September, 1967, the patient returned to the osteopath, who informed him that his wife's pregnancy had been caused by sperm trapped in the seminal tract above the area of the incision. He told the patient that there were no live sperm in his semen.

In July, 1968, the wife again found that she was pregnant. The osteopath again tested the husband and told him he was sterile. In February, 1969, the wife gave birth to a normal child.

In May, 1969, the patient and his wife brought action against the osteopath, contending that he was guilty of negligence, breach of implied warranty, and misrepresentation. The trial court entered judgment for the osteopath.

On appeal, the court found that the action was barred by the two-year statute of limitations, which began to run when the cause of action accrued. The husband and wife contended that the osteopath fraudulently concealed the existence of the cause of action and that the statute of limitations began to run when they discovered it. The appellate court affirmed the judgment of the trial court, finding that the husband and wife had learned of the third pregnancy about a year before expiration of the statute of limitations but had waited 18 months before filing suit.

The supreme court held that in malpractice cases arising from vasectomies, the statute of limitations began to run on the date of discovery of the facts concerning the failure of the operation or from the date the facts should have been discovered with ex-

ercise of ordinary care and diligence. The court pointed out that if the limitation period was measured from the date of the operation and the discovery of fertility was not made until the period of limitations had run, legal remedy would not be available to the injured party. The supreme court therefore reversed the judgments of the lower courts and sent the case back for trial.—*Hays v. Hall,* 488 S.W.2d 412 (Tex.Sup.Ct., Dec. 13, 1972; rehearing denied, Jan. 17, 1973).

Editor's Note: A prior decision was reported in *The Citation,* Vol. 25, No. 6, p. 89.

Functional Obstructive Cardiomyopathy

(Continued from page 338)

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Thoracic Outlet Relief

(Continued from page 331)

decompensation or vascular complications, excellent results will be obtained.

First rib resection by the transaxillary approach is, in our opinion, the treatment of choice for compression at the thoracic outlet, since the first rib is the only common denominator for all the compressive areas.

Summary

A prospective study utilizing transaxillary first rib resection for treatment of the thoracic outlet compressive syndrome has been presented.

One hundred thirty-three patients met the criteria of demonstrable vascular compression with nerve loss prior to operation. Two patients were operated upon for severe vascular compression without nerve decompensation. All patients gained relief of neurovascular compression. There was no mortality. There were eight operative complications. Using these preoperative criteria, excellent results can be gained from this form of therapy.

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Vox Dox

Vox Dox Editor:

I have read with interest Dr. William Roy's column, *D. C. Line.* Dr. Roy seems to be trying hard to be Good Ole Bill, Friend of All, Brother to the Doctor, Defender of the Poor, and Upholder of the Flag, Apple Pie, and Mom. However, to a lot of us, he comes across like a politician. Moreover, he sounds increasingly like a left-wing Kennedy politician.

Apparently, his entire political attitude revolves around two premises: (1) Some sort of "Medical Crisis" exists in the United States; (2) The only possible answer is more and more federal control. Neither of these premises is necessarily true.

I almost gagged over the glorious tale of how marvelous the practice of medicine is under the socialized Canadian system. If "Good Ole Bill" thinks it's so great, maybe he'd like to go there and stay. I'll buy his ticket!

At very least, I think you should give equal space each month to someone who could adequately present the opposing political view. I resent the use of my state medical publication as a springboard for the political aspirations of "Good Ole Bill."

Sincerely,

Donald C. Coldsmith, M.D.

AMA Committee on Community Health Care

Statement on Health Outreach

The concept of health outreach has gained wide acceptance by a number of community health programs throughout the nation's rural and urban poverty areas. The outreach worker (known by many titles, such as health aide, health advocate, community aide, community representative, health agent, family health worker, health street worker, and ombudsman) is generally a resident of the area served and is employed to (1) bridge the cultural gap between patients, professional staff, and the community, (2) improve communications between these groups, and (3) assist in the effective delivery of health care to patients and their families. The outreach worker is an important component of the health care team.

Health outreach workers usually work under the supervision and coordination of a professional staff member of the health team. They are being trained to perform a variety of activities based upon their capabilities and the existing needs of the community. Some of these activities are: (a) providing information on health and health-related problems including case finding in the community; (b) informing the community residents of available medical and health services; (c) assisting in the development and use of health educational materials in language and cultural context that is understood and accepted by the people; (d) helping to demonstrate how to prepare and serve nutritious meals using acceptable and economical foods; (e) assisting the patient in following the therapeutic regime recommended; (f) alerting the professional staff to family health or health-related problems; (g) conducting follow-up visits in homes; and (h) participating in community and health care team discussions.

At the 1971 Clinical Convention, the House of Delegates adopted Council on Medical Service Report B entitled "Community Health Delivery Programs." This report was prepared as a result of the task force site visits by the Committee on Community Health Care to more than 30 different community health programs. The observations regarding outreach workers were:

> "Community health workers were utilized in all but four programs although they were known by a range of titles, such as health agents, family workers, and outreach workers. These allied health personnel served as paid staff members of the center and performed many functions, including health education, direct patient care, and patient assistance, such as getting the patient to and from the center and the hospital and in patient follow-up. One of their most important functions was serving as "ombudsman" or the primary interface in relating the health program to the community and the community to the health program. When well organized, this function was observed to make some programs more successful than others from the standpoint of community use and acceptance. The development and use of the health outreach concept has been one of the most significant and meaningful aspects of community health programs. Besides expediting the flow of people into the health care delivery system, it has provided opportunities for jobs and career progression and given these workers a sense of participation and involvement in their program. In addition, it has contributed to the overall economic upgrading of the individual residents of the total community."

The report also concluded that one of the key elements to successful community health programs was the "utilization of the health care team approach, including full use of community outreach workers."

The Council on Medical Service and its Committee on Health Care of the Poor and Committee on Community Health Care believe there are a number of sound reasons that justify the utilization of outreach workers, particularly in poverty areas. Among the reasons are that the use of outreach workers: (1) tends to enhance professional standards of practice since such personnel can free physicians, nurses, dentists, and other health professionals to better utilize the time for which they are trained and extend the scope of services to a larger patient population; (2) provides an additional source of manpower to meet community needs, especially in those areas where there is a shortage of professional health staff; (3) obviates many of the traditional problems of understanding and communication in getting health services to those in need; (4) assists the professional staff in becoming more responsive and accountable to the community served; and (5) provides meaningful jobs and, as a result, benefits the community economically and socially.

To improve community acceptance of health programs and access to health care services, the Council on Medical Service submits the following recommendations:

1. The AMA and state and local medical societies should encourage use of health outreach

personnel in all appropriate community health care delivery settings.

2. The AMA should gather data concerning more effective utilization of outreach workers. This should include evaluation of existing training programs and subsequent recommendations for improvements.
3. The AMA should institute educational activities for physicians and other health professionals concerning the utilization of outreach workers.

Change of Address

Please notify the Kansas Medical Society of any changes in address.

Help keep the mailing list up to date

Disability Insurance

(Continued from page 344)

set of findings in the criteria. (This is the only way the widow 50 or over can qualify for disability benefits.) However, for all other claimants whose impairments fall short of this test, such factors as age, education, and work experience added to the functional limitations imposed by the medical condition are taken into consideration in making the disability decision.

The complete criteria, including the medical findings listed by body system, are contained in a handbook designed especially for professionals who come in contact with the disabled population. The handbook describes impairments in terms of specific symptoms, signs, and laboratory findings which are presumed to be severe enough to prevent a person from working for a year or longer.

The handbook may be obtained from Disability Determinations Unit, Post Office Box 5057, Topeka, Kansas 66605. This office also welcomes any inquiries from physicians who wish to know more about the Social Security disability program and its policies and procedures.

115th Annual Session

KANSAS MEDICAL SOCIETY

May 5-8, 1974, Topeka

MODERN ASPECTS OF TRAUMA

1. **Aeromedical Transportation of the Injured**
2. **Medico-Legal Aspects of Trauma**
3. **Symposium on Fat Embolism**

The 1974 meeting will be held in conjunction with the following specialty societies:

KANSAS SOCIETY OF PATHOLOGISTS
KANSAS OBSTETRICAL AND GYNECOLOGICAL SOCIETY
KANSAS ALLERGY SOCIETY
KANSAS SECTION ON EAR, NOSE AND THROAT
KANSAS SOCIETY OF ANESTHESIOLOGISTS

Personalities—IN KANSAS MEDICINE

The new officers of the Kansas Chapter, American Academy of Family Physicians are as follows: **Richard R. Brummett**, Neodesha, President; **Alex C. Mitchell**, Lawrence, President-Elect; **Philip H. Hostetter**, Manhattan, Vice President; **Anol W. Beahm**, Great Bend, Secretary; **Lawrence E. Leigh**, Overland Park, and **Ernie J. Chaney**, Belleville, Delegates; **Donald D. Goering**, Salina, and **Kenneth L. Lohmeyer**, Emporia, Alternate Delegates; **Floyd L. Smith**, Colby, to the Board of Directors.

E. C. Bryan, Erie, was honored by the community for the 40 years of medical practice he had given to the area.

Dean M. Miller is moving his practice from Topeka to Parsons.

After 47 years of medical practice in Washington, **D. A. Bitzer** is retiring.

William T. Collier, McPherson, attended the annual meeting of the Flying Physicians Association, in San Diego.

Included in the 1973-74 edition of *Who's Who Among American Women* was **Agnes J. Aves**, Parsons.

George S. Bascom, Manhattan, was the principal speaker at the graduation exercises for the Practical Nursing School.

P. E. Beauchamp, Sterling, has announced his retirement from practice effective October 1. He has practiced in the same area since 1936.

NEW MEMBERS

The JOURNAL *takes this opportunity to welcome these new members into the Kansas Medical Society.*

J. Barry Claycomb, M.D.
P. O. Box 829
Topeka, Kansas 66601

Burton A. Dudding, M.D.
K. U. M. C.
Kansas City, Kansas 66103

Joseph J. Duerksen, M.D.
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Kansas City, Kansas 66103

Martin S. Hirsch, M.D.
10343 Maple
Shawnee Mission, Kansas 66207

William R. Jewell, M.D.
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3000 West 50th St.
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RADIATION PROTECTION STANDARDS by Lauriston S. Taylor. C.R.C. Press, Cleveland. 1971. 110 pages. $11.50

This monograph deals primarily with the history of radiation protection, beginning in 1925, and progressing to the last revision in 1972. The monograph provides an excellent chronological account of the International Commission on Radiological Protection and National Council on Radiation Protection, from the early days, up to the present time when there were enough foresighted leaders to establish tolerance doses, maximal permissable dose, and dose limiting recommendations to make the public as well as the users radiation conscious that radiation protection is essential regardless of the source of ionizing radiation being used.

Dr. Taylor has done an excellent job in summarizing this progress and logic of many of these recommendations. His concise style of writing makes for easy reading.

This is recommended reading for any one that is interested in the history of ionizing radiation, protection and current status of recommendations for protection. There is an excellent bibliography accompanying the monograph.—*R.C.L.*

HANDBOOK FOR THE ORTHOPEDIC ASSISTANT by F. Richard Schneider. The C. V. Mosby Company, St. Louis. 1971. 198 pages, 177 illustrations. $10.75.

F. Richard Schneider, M.D., has authored this excellent aid to the orthopedic assistant working under the supervision of an orthopedic surgeon.

Orthopedic terminology is increased as the book leads one through the musculoskeletal system, bone development, joints and fractures. The text then explains basic principles and techniques in the treatment of trauma, with instruction in applying plaster casts and, when necessary, skin and skeletal traction.

From a non-professional standpoint, I feel this textbook would be of extreme value not only as an instructional manual but also an excellent reference source for the experienced orthopedic assistant.—*C.M.*

RESPIRATORY PHYSIOLOGY by N. Balfour Slonim and Lyle H Hamilton. The C. V. Mosby Company, St. Louis. 1971. 119 pages. $10.75.

The introduction to this book starts with the broad definition as the science of processes and function of living biologic systems. The introduction discusses the definition of a few basic terms such as pulmonary ventilation, pulmonary circulation, ventilation/perfusion distribution, diffusion, transport of gases and regulation of respiration. It also includes some discussion of learning for the medical student, inhalation therapy student and the physician. A short description of the history of respiratory physiology is included.

The book is well written and covers the subject matter quite well. There are several chapters which are covered quite well in other books, but are also necessary for this book such as functional anatomy of the bronchopulmonary system, laws describing the behavior of gases, lung volume and its subdivisions. However, these chapters are well written and contribute a vital part to this book especially for its use as a teaching book. The chapters on respiratory physiology of the newborn and on respiratory responses to unusual environments and special atmospheres were especially well presented.

The appendix contains many useful tables and the glossary has the usual well presented definitions.

This is one of the better books I have read on respiratory physiology and feel it certainly belongs in every medical library, inhalation therapy school and inhalation therapy department.—*W.N.*

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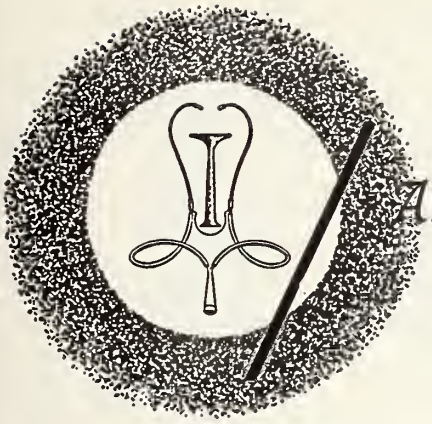

Announcements

Professional meetings, conferences, and postgraduate courses of national importance are listed for the* Doctor's Calendar. *Notice of the session is posted in advance to allow the physician time to make preparations.

SEPTEMBER

Sep. 17-18 AMA Congress on Occupational Health, 33rd Annual. Benjamin Franklin Hotel, Philadelphia.

Sep. 19-21 Colorado Medical Society, Annual Meeting. Broadmoor Hotel, Colorado Springs.

Sep. 20 American Society of Internal Medicine, Interim Meeting, Dallas. Write: W. R. Ramsey, 525 Hearst Bldg., San Francisco 94103.

Sep. 27-29 Cancer of the Colon and Rectum, American Cancer Society 2nd National Conference. Americana Hotel, Bal Harbour, Florida. Write: S. L. Arje, M.D., 219 East 42nd, New York 10017.

Sept. 29 College of American Pathologists & Bethesda Naval Hospital. *Sero-Diagnosis of Cancer Symposium.* Write: CAP, 1775 K St., N.W., Washington, D. C. 20006.

OCTOBER

Oct. 1-4 Academy of Family Physicians, Denver. Write: Roger Tusken, Volker at Brookside, Kansas City, Missouri 64112.

Oct. 5-6 Kansas Heart Association Annual Meeting, Hutchinson. Write: Bill Stanley, 5229 W. 7th, Topeka, Kansas 66606.

Oct. 7-11 AACHP Third Annual, Kansas City. Write: R. R. Solee, Comprehensive Health Planning, 535 Kansas Ave., Topeka, Kansas 66603.

Oct. 15-19 American College of Surgeons, Chicago. Write: C. R. Hanlon, M.D., 55 E. Erie, Chicago 60611.

Oct. 20-25 American Academy of Pediatrics, Chicago. Write: R. G. Frazier, M.D., 1801 Hinman, Evanston, Illinois 60201.

Oct. 20-26 Annual Otolaryngologic Assembly, University of Illinois Hospital. Write: Otolaryngology, PO Box 6998, Chicago 60680.

Oct. 21-25 American College of Chest Physicians, Annual Scientific Assembly, Four Seasons Sheraton Hotel, Toronto. Write: ACCP, 112 E. Chestnut, Chicago 60611.

Oct. 25-27 American College of Gastroenterology, Annual Postgraduate Course, Biltmore Hotel, Los Angeles. Write: ACG, 299 Broadway, New York 10007

Oct. 29-31 Omaha Mid-West Clinical Society, 41st Annual Postgraduate Assembly, Hilton Hotel, Omaha. Write: J. A. Haggstrom, M.D., 1040 Medical Arts Bldg., Omaha 68102.

Oct. 29-Nov. 1 Interstate Postgraduate Medical Association, Palmer House, Chicago. Write: A. Ochsner, M.D., P.O. Box 5445, Madison, Wisconsin 53705.

NOVEMBER

Nov. 2-3 20th Western Cardiac Conference, Cosmopolitan Hotel, Denver. Write: Colorado Heart Association, 1375 Delaware St., Denver 80204.

Nov. 14-17 Automotive Medicine 17th Annual Conference, Hilton Inn West, Oklahoma City. Write: Page Waller, Oklahoma State Department of Health, NE 10th & Stonewall, Oklahoma City 73105.

Nov. 15 Therapy in the Rheumatic Diseases; 4th Annual Arthritis Symposium, St. Francis Hospital Nurses Auditorium, Wichita. Write: Frederick Wolfe, M.D., Chairman, Professional Education Committee, Kansas Chapter Arthritis Foundation, 345 N. Hillside, Wichita 67214.

Nov. 18 Kansas Medical Society House of Delegates, University Ramada Inn, Manhattan.

Nov. 29-Dec.1 Virology and Immunology in Human Cancer, Waldorf-Astoria Hotel, New York. Write: S. L. Arje, M.D., American Cancer Society, 219 East 42nd St., New York 10017.

University of Colorado:

Sept. 24-28	*Hospital Medical Staff Conference*
Oct. 8-12	*High Risk Infant Care*
Oct. 14-18	*Pathology in Gynecology and Obstetrics*
Oct. 15	*Oral Cancer Seminar*

For further information, write the Office of Postgraduate Medical Education, University of Colorado School of Medicine, 4200 E. Ninth Ave., Denver 80220.

Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high-contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

Influenza Vaccine Recommendations

For use during Fall 1973, two new and distinct influenza vaccines are expected to be commercially available. The first will be a bivalent preparation, formulated with antigens representative of the most recently prevalent type A "England" and type B "Massachusetts" strains. This will be the first vaccine formulated to specifically protect against the type A "England" strain which was first recognized during the winter of 1972-73. The type B strain in this bivalent formulation is one which has been prevalent since 1971.

The second vaccine to become available will be formulated to protect against a type B strain which was first recognized in Hong Kong in December 1972, and was subsequently associated with influenza outbreaks in Australia and England. It is too early to judge whether this strain will generally supplant the type B strain of the bivalent vaccine formulation in the coming influenza season. However, it is reasonable to expect that this newer "B" strain will become more widely disseminated. Accordingly, the new type B influenza vaccine should be used as a supplemental vaccine for optimal protection of persons at high-risk who are already recommended to receive the bivalent vaccine. Annual influenza vaccination is recommended for persons of all ages who have such chronic conditions as (1) heart disease of any etiology; (2) chronic bronchopulmonary diseases; and (3) diabetes mellitus and other chronic metabolic disorders. Annual vaccination also is recommended for older persons because influenza outbreaks are commonly associated with excess mortality in older age groups.

The primary series of bivalent influenza vaccine has traditionally been two doses. Preliminary data indicate that with the more potent influenza vaccines available in recent years, the second dose provides little additional benefit. It is, therefore, reasonable to give a single dose of vaccine for either primary or annual booster vaccination.

A single dose of the supplemental monovalent type B vaccine should follow and not be given simultaneously with the bivalent vaccine. This is because the additional amount of antigen in the monovalent product might increase the chance of adverse reactions. Furthermore, separating the vaccines by two weeks or more might enhance an overall type B antibody response. Influenza vaccination should be scheduled for completion by mid-November.

As in the past, the influenza vaccines are prepared from viruses grown in embryonated eggs and ordinarily should not be administered to persons clearly hypersensitive to egg protein, ingested or injected.

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THE Journal OF THE Kansas Medical Society

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Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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Volume LXXIV, Number 10—October, 1973

The JOURNAL of the KANSAS MEDICAL SOCIETY

Contents for October

Scientific Articles

Non-Operative Visualization: Per-Oral Transduodenal Pancreatography and Cholangiography—Giomar Gonzalez, M.D. and Alonso Galvan, M.D., Kansas City, Kansas . 361
Physicians and Psychiatry: Physicians' Attitudes Toward Psychiatry—John D. Pro, M.D. and Don Fortin, M.D., Kansas City, Kansas 367
Pericardial Cyst: 500-Gram, 15 x 8 x 8 Cm.—Jose M. Vera-Roman, M.D. and Don R. Miller, M.D., Kansas City, Kansas 369

KUMC Trauma Conference

Multiple Stab Wounds of the Head, Neck, and Thorax—Edited by Arlo S. Hermreck, M.D. and Frederick W. Reckling, M.D., Kansas City, Kansas 371

Medical History

Isaac Arthur Abt, 1867-1955—Ronald D. Greenwood, M.D., Boston Mass. 376

Miscellaneous

Advertisers 27
AMA: Statement on Venereal Diseases 15
Announcements 6
Book Reviews 20
Classified Advertisements 27
Council Minutes 388
D. C. Line 392
Editorial Comment 384
House of Delegates, Resolutions 389
Information for Authors 366
Kansas Morbidity Incidence Report 22, 24
Kansas Press Looks at Medicine 25
KUMC Postgraduate Education 382
Medical-Legal Page 387
Month in Washington 8
Obituaries 390
Personalities 391
President's Message 383
Surgical Asepsis 18
Woman's Auxiliary 386

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Scientific ARTICLES

Non-Operative Visualization

Per-Oral Transduodenal Pancreatography and Cholangiography

GIOMAR GONZALEZ, M.D.* *and*
ALONSO GALVAN, M.D.,† *Kansas City, Kansas*

A diagnostic procedure which allows the non-operative visualization of the pancreatic, hepatic, and intrahepatic ducts. It can be performed successfully in the large majority of patients. The presence of obstruction of the pancreatic duct, either complete or incomplete, can be easily demonstrated. Strictures, obstructions, and displacement of the hepatic and intrahepatic ducts are also well demonstrated by this method.

THE AVAILABILITY of the fibro-optic instruments permits the mucosal surfaces of the gastrointestinal tract to be visualized directly. This procedure has improved the diagnostic accuracy of lesions in the esophagus, stomach and duodenum,[1] and with the use of the duodenoscope the endoscopist is able to inspect the duodenal papilla and to catheterize the ampulla of Vater and the common duct. The injection of contrast material under direct fluoroscopic control with appropriate spot and overhead films permits the evaluation of the pancreatic duct and some of its branches, as well as visualization of the common and hepatic bile ducts and some of the intrahepatic ducts.

The roentgenographic evaluation of diseases of the pancreas includes the routine upper gastrointestinal series, hypotonic duodenography, selective and superselective arteriography, and pancreatic scanning. The criteria for diagnosis of the different disease processes as well as the application of each one of these procedures have been recently reviewed.[2] The introduction of ultrasound as a diagnostic tool for evalution of intra-abdominal and retroperitoneal masses has added a new dimension in the study of the pancreas.[3, 4]

The use of pancreatography as a diagnostic procedure for diseases of the pancreas is well documented in the literature.[5-10] However, most of the experience acquired in the performance and interpretation of pancreatography has been obtained by means of operative injection of contrast material into the pancreatic duct, either following duodenotomy and cannulation of the ampulla of Vater or by cannulation of the duct following resection of the tail of the pancreas.

In 1965, Rabinov and Simon[11] performed the first non-operative pancreatogram. Using a per-oral flexible tube containing a cannula, they were able to catheterize the pancreatic duct under fluoroscopic control but without the benefit of direct visualization of the ampulla of Vater.

Non-operative visualization of the hepatic and common bile ducts can be accomplished by intravenous cholangiography,[12, 13] transhepatic percutaneous

* Diagnostic Radiology, University of Kansas Medical Center, Kansas City, Kansas 66103.
† Gastroenterology, Department of Internal Medicine, University of Kansas Medical Center, Kansas City, Kansas 66103.

cholangiography,[14] and transjugular cholangiography.[15] However, each of these methods has disadvantages and technical difficulties. They carry a certain morbidity, and there still exist some problems concerning the demonstration of the integrity of the intrahepatic and common bile ducts which challenge the radiologist.

During the past year, we have performed per-oral transduodenal pancreatography and cholangiography following the cannulation of the duodenal papilla and the ampulla of Vater. Eighteen patients have been studied. The technical aspects of the cannulation of the ampulla, as well as the roentgenographic manifestations of pancreatic disease, will be described and illustrated. The advantages of retrograde cholangiography performed with this method over other methods of visualization of the hepatic ducts will be emphasized and demonstrated.

Technique

The patient is premedicated with an intramuscular injection of 10 to 15 mg diazepam and 1 mg atropine 30 minutes prior to the procedure. The patient should be well hydrated. During the procedure, intravenous normal saline is given, since intermittent intravenous administration of diazepam and propantheline bromide is needed throughout the procedure to keep the patient comfortable and to diminish duodenal motility.

The gastroduodenoscope *(Figure 1)* is a side-view endoscope 1 cm in diameter and 110 cm long. It has channels for biopsy, air insufflation, suction, water irrigation, and cannulation. It is flexible and well tolerated by the patient. The viewing field is 70° and extends from 20 mm to infinity. The first part of the endoscope is introduced while the patient is lying in the left lateral decubitus position, and is guided with the middle and index fingers of the left hand toward the center of the pharynx. The scope is gently pushed while the patient swallows, and is subsequently passed into the stomach.

In the stomach, the antrum is localized and the pylorus is surveyed for abnormalities which might hamper passage into the duodenum. Since the endoscope is a side-viewing instrument, the relationship of the pylorus to the tip of the scope must be kept in mind. The tip of the scope is placed as close as possible to the pylorus, with the scope in upward flexion and keeping the superior portion of the pyloric ring in the field of view at all times. The endoscope is then gently introduced into the duodenum.

Within the duodenal bulb the endoscope lens will be directed upwards. A clockwise turn of 90° is necessary to continue the introduction of the scope into the duodenum. Going through the first and second portions of the duodenum is the most difficult maneuver. The scope should not be advanced when one has a complete view of the lumen of the duodenum, but should be deflexed or placed in a position where only one of the walls of the duodenum is visualized. This is usually the postero-inferior wall.

The duodenal papilla *(Figure 2)* is located in the postero-medial wall of the descending portion of the duodenum about 2-4 cm from the superior duodenal angle. For cannulation purposes it is absolutely necessary to place the tip of the endoscope directly underneath the papilla and identify the orifice, so that the cannula can be introduced into it. If the orifice cannot be readily seen, gentle trial and error manipulation at the apex of the papilla is performed, using the flexion and deflexion controls or the right

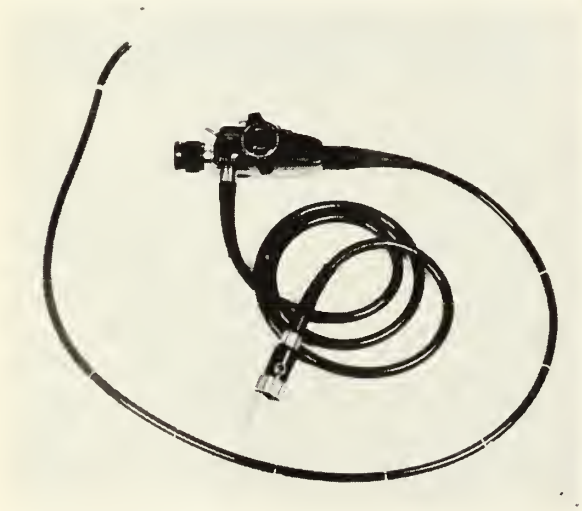

Figure 1. The Olympus Duodenofiberscope. A photographic camera may be attached for color photography.

Figure 2. Duodenal papilla being cannulated.

and left controls. The latter add leverage for the introduction of the cannula into the orifice.

It is necessary to suppress duodenal peristalsis for successful cannulation. This is accomplished with 30-60 mg of intravenous Propantheline-Bromide. Two or three milliliters of meglumine diatrizoate 60 per cent diluted with equal parts of saline to produce an iodine concentration of 14.5 per cent is used as contrast material. In our experience, the average time spent in the fluoroscopic room has been 45-60 minutes. This time includes the introduction of the duodenoscope, the identification of the duodenal papilla, the cannulation of the ampulla, and filling of the ducts with contrast material. The fluoroscopic time is not more than 10 minutes, and fluoroscopy is only needed to demonstrate the presence of contrast material in the ducts.

Pancreatic Disease

Case One

A 62-year-old female was admitted to the University of Kansas Medical Center with the chief complaint of abdominal pain and weight loss of eight months duration. The pain was originally located in the epigastrium but later migrated to the lower abdomen and low back. It was aggravated by eating. Review of symptoms was negative. Physical examination was essentially within normal limits with no organomegaly or masses palpated.

The upper gastrointestinal examination *(Figure 3A)* revealed displacement of the stomach anteriorly by a retroperitoneal mass. There was a suggestion of calcifications in the region of the head of the pancreas.

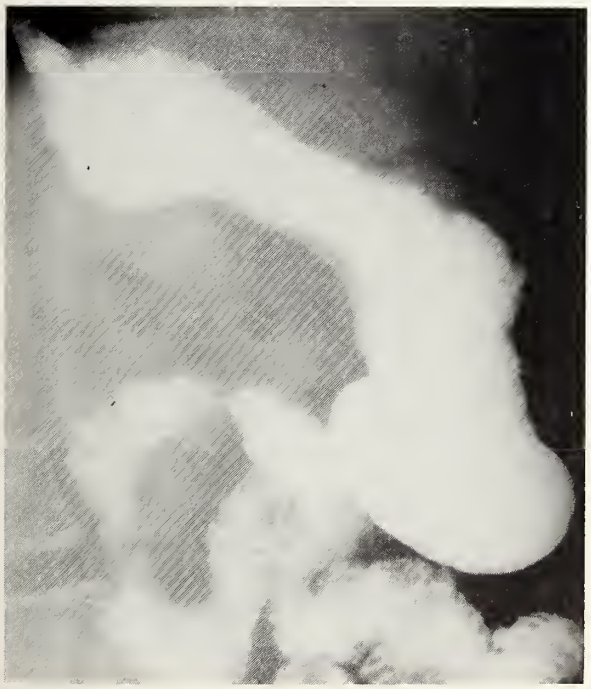

Figure 3A. Lateral view of the stomach showing extrinsic pressure effect on the posterior wall by a mass in the body of the pancreas.

Figure 3B. The distal segment of the pancreatic duct is demonstrated along with numerous secondary branches.

A fibroduodenoscope was passed into the duodenum and injection of contrast material was made into the pancreatic duct. Early during the injection, partial obstruction of the main duct was evident with filling of secondary radicles *(Figure 3B)*. Later, the whole length of the duct was demonstrated *(Figure 3C)*. Carcinoma of the pancreas was suspected.

During laparotomy, a 6 cm mass was found in the body of the pancreas which proved to be an adenocarcinoma. Two palpable liver nodules proved to be metastatic disease.

Figure 3C. The complete pancreatic duct. Irregularities in the contour are evident (arrow).

Comment

Although complete obstruction to the retrograde flow of contrast material has been described as a roentgenographic finding of carcinoma of the pancreas,[6] this case illustrates carcinoma producing only partial ductal obstruction. There are irregularities along the contour of the duct at the precise level where the tumor was found at laparotomy *(Figure 3C arrow)*. This finding indicated that the tumor had already invaded the duct. Displacement of the branches of the main pancreatic duct may not be a reliable sign of a mass since secondary branches are frequently not filled with contrast material. Criteria for the diagnosis of carcinoma of the pancreas by this modality have not as yet been completely documented.

Case Two

A 49-year-old male was admitted to the University of Kansas Medical Center with the chief complaint of abdominal pain. The patient had recurrent episodes of acute pancreatitis over the last 2½ years associated with alcoholic intake. Cholecystectomy had been performed because of stones 11 years prior to this admission. He had lost 13.6 kg (30 lbs) during the last year. Physical examination was noncontributory.

Figure 4A. Extensive calcifications in pancreas. The large stone (arrow) appears to be located in the head of the organ.

Figure 4B. The hepatic duct shows extrinsic pressure from the enlarged head of the pancreas.

Laboratory examination showed a slight abnormality in the glucose tolerance test. Pancreatic enzymes were within normal limits.

A plain film of the abdomen *(Figure 4A)* revealed calcifications in the body of the pancreas and a large stone *(arrow)* in the region of the pancreatic head.

A fibroduodenoscope was introduced into the duodenum, the papilla was cannulated, and contrast material was injected into the common duct. The hepatic duct was displaced by extrinsic pressure from the enlarged head of the pancreas *(Figure 4B)*. A short segment of the pancreatic duct was visualized with demonstration of complete obstruction to the retrograde flow by the previously noted calcified mass *(Figure 4C, arrows)*. No contrast material was visualized beyond the obstruction.

Comment

The role of pancreatic duct obstruction as a cause of chronic pancreatitis has been documented in the literature.[9] The site of the obstruction can be at the level of the ampulla of Vater or within the main duct. Although in the chronic stage of this disease the response to surgical treatment is somewhat less than satisfactory,[9] the demonstration of mechanical obstruction to the free flow of pancreatic secretions may justify a surgical procedure in an attempt to restore the normal passage of pancreatic juice. Howard[6] reported that it is more common to find partial obstruction of the pancreatic duct, rather than complete obstruction in cases of chronic relapsing pancreatitis.

Figure 4C. Complete obstruction to the retrograde flow by the large stone (arrows).

Retrograde Cholangiography

Case One

This 49-year-old male was admitted to the University of Kansas Medical Center with jaundice. One month prior to admission he developed malaise, belching, and anorexia. He soon became jaundiced with clay-colored stools and dark urine. He denied abdominal pain, blood transfusions, or use of medication. No history of alcohol intake was found. Weight loss during the past month amounted to 6.7 kg (15 lbs). Physical examination revealed jaundiced sclera and slight hepatomegaly. Pertinent laboratory findings on admission included the following: total bilirubin, 16.6 mg/100 ml, with direct of 9.9 mg/100 ml; alkaline phosphatases, 18.6 Bessey Lowry units/100 ml; and serum glutamic oxaloacetic-acid transminase (SGOT), 60 units. Urinalysis showed 3+ bilirubin.

The injection of contrast material into the pancreatic duct demonstrated a normal course and no obstruction *(Figure 5, arrow)*. The injection of contrast material into the hepatic duct showed complete obstruction to the retrograde flow by a smooth surfaced mass which produced the characteristic meniscus appearance of an impacted stone *(Figure 5, arrow)*. At laparotomy, a grossly dilated proximal segment of the hepatic duct was found with impaction of a large stone in the distal end of the common bile duct.

Comment

The differential diagnosis between hepatocellular and obstructive jaundice is still difficult. The demonstration and evaluation of the intrahepatic, hepatic, and common bile ducts prior to laparotomy has been accomplished in the last decade with the use of in-

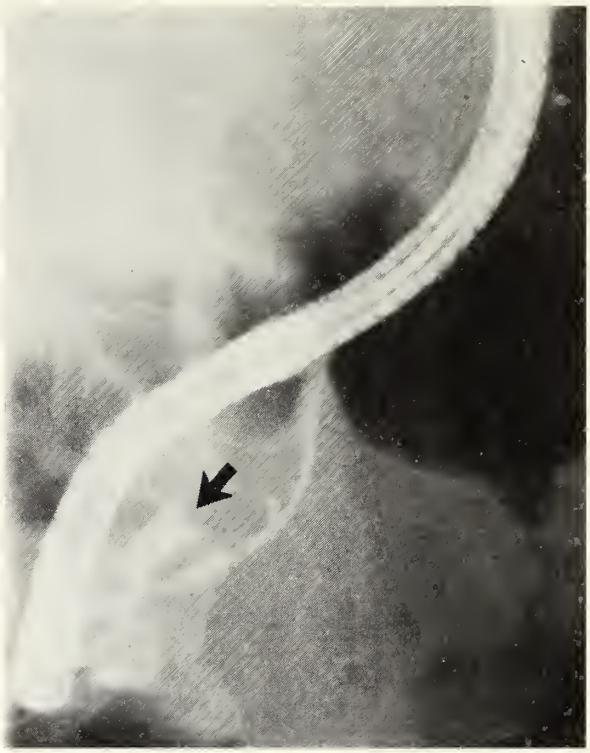

Figure 5. Obstruction of the hepatic duct by an impacted radiolucent stone.

travenous cholangiography, transhepatic cholangiography, and transjugular cholangiography. Intravenous cholangiography is a routine procedure with a high degree of diagnostic accuracy.[12, 13] However, it requires fairly normal liver function. Therefore, in patients with jaundice, the use of intravenous cholangiography is limited. Transhepatic and transjugular cholangiography are being used in some medical centers with good success.[14, 15] The risk of producing bile peritonitis and hemobilia is present as well as the possibility of the development of infection.

Transduodenal cholangiography demonstrates to a large extent the anatomy of the ducts, the presence of strictures or obstructions, and displacement. If complete obstruction is encountered, only the distal segment of the common duct will be demonstrated. However, the roentgenographic characteristics of the obstruction should allow the correct diagnosis to be made.

Discussion

The direct visualization of the duodenal papilla with retrograde catheterization of the ampulla of Vater, pancreatic and bile ducts, and injection of contrast material into these structures is a diagnostic procedure which can be performed with minimal trauma to the patient. Complications to the performance of this procedure are related to the introduction of the fibroduodenoscope into the duodenum and are mini-

mal with the newer flexible instruments. The possibility of chemical pancreatitis must always be considered following introduction of contrast material into the pancreatic duct. However, reports in the literature[7, 16, 17] and our own experience have shown that following pancreatography only minimal changes in the enzymes are produced. In our experience only one patient developed acute pancreatitis, which resolved without sequela.

The information obtained by this method increased the accuracy of the preoperative diagnosis and, therefore, improved the management of patients with suspected pancreatic or biliary tract disease. The success of cannulation of the structures is directly related to the experience of the endoscopist. Our early experience was slightly disappointing. In three of our first six patients we were not able to introduce the cannulating catheter. However, since then we have been able to cannulate the duct in 15 patients.

Per-oral transduodenal pancreatography is indicated in patients with suspected disease of the pancreas including chronic relapsing pancreatitis, pseudocyst, retention cyst, and carcinoma. Retrograde visualization of the common and intrahepatic bile ducts offers an alternative diagnostic procedure in patients in which the diagnosis of obstructive jaundice is entertained.

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(Continued on page 382)

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Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

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Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

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Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

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All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

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Physicians and Psychiatry

Physicians' Attitudes Toward Psychiatry

JOHN D. PRO, M.D. *and* **DON FORTIN, M.D.**, *Kansas City, Kansas*

PRACTICING PHYSICIANS are frequently the first resource turned to in emotional crises; yet, Hyams, *et al.*[1] and Liddon[2] have shown that physicians have very little time to devote to these emotional needs. In our own experience at a large medical center, where psychiatric referral and consultation are common, we have become aware of this problem, as well as the inadequate relationship between the primary physician and the psychiatrist. Consequently, we became interested in assessing the status of psychiatry and psychiatric referral from the standpoint of physicians practicing in Kansas. An understanding of these attitudes and referral practices may enable those who deal with emotional illness—either practicing physicians, psychiatrists, or allied professionals—to more clearly define the problems they face and to deliver more adequate mental health care.

Method

A three-page questionnaire containing 16 multiple choice and seven general information questions was sent to a random sample of physicians drawn from the current membership roster of the Kansas Medical Society.

The questionnaire contained 23 items which concerned four broad categories: general background information about the physician and his practice; attitudes toward psychiatry; practices of referral to psychiatrists; and attitudes toward mental illness. The physician was allowed to define "emotional illness," since many conceptions of this phrase were expected and since no pilot study was done to gather a consensus definition. A basic understanding and appreciation for a general medical model of emotional disorders was assumed.

Results

Of the 200 questionnaires sent, 52 per cent were returned; of these, 11 were invalidated because the respondents had either retired or were psychiatrists themselves. Questionnaires in a few cases were partially completed; they were disregarded for any Chi-square analysis. The total number of physicians answering each question was calculated. Also, the per cent answering each choice under the questions was determined. The Chi-square statistical method was used to delineate relationships between various aspects of the data.

Of the respondents, 38 per cent were general practitioners and 62 per cent were specialists. The mode of the year graduated from medical school was 1950. Most of those responding (71 per cent) were situated less than ten miles from a psychotherapist; 40 per cent practiced in a town with population over 100,000, while 21 per cent practiced in an area of less than 5,000 population.

With a 52 per cent response in a questionnaire survey of Kansas physicians, most referred less than 10 per cent of their patients to psychiatrists, despite an accepted much higher rate of emotional disorders in their practice. Some of the reasons of this discrepancy are explained.

Most of the respondents (74%) regarded the psychiatrist's intervention "always" or "usually" helpful, and most of them "always" or "usually" felt that his communication was prompt (65%), and useful and pertinent (79%).

Sixty-seven per cent of the physicians felt that a psychotherapist should be a physician; 14 per cent stated he should be a non-physician; 20 per cent felt either would do. Forty-four per cent agreed that psychiatry was changing toward the mainstream of medicine; 34 per cent disagreed, stating that psychiatry was changing toward humanism and the social sciences. A large number (23%) responded with "other" to this issue, and indicated either an ambiguity in the question, or that there was a combined tendency toward medicine and the social sciences. Seventy-two per cent of the respondents expressed a greater need for psychiatrists, and 56 per cent found the classical diagnostic categories of emotional illness understandable.

The majority (67%) of the respondents reported a referral rate of less than 10 per cent of their patients who manifested symptoms of emotional illness. On the other hand, 75 per cent stated that more

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than 10 per cent of all their patients did have symptoms of emotional illness (40% reported that 10-30% of their patients had emotional symptoms). Thirty-four per cent indicated that they go further into the patient's problem to determine the need for psychiatric referral, and another 34 per cent evaluate the significance of the problem and initiate a mode of therapy themselves.

No statistically significant relationships could be established between the percentage of patients referred and population of the town, distance to a psychiatrist, type of physician (general practitioner or specialist), or year graduated. However, the Chi-square was significant at the 0.05 level for the relationship between the year graduated from medical school and the type of therapist who should treat emotional illness (physician, non-physician, either). That is, a significant number of physicians graduating after 1950 acknowledged that the psychotherapist could either be a physician or a non-physician; whereas, those graduating before 1950, felt the therapist must be a physician.

Discussion

Most of the physicians reported a referral rate to psychiatrists of less than 10 per cent, regardless of the year they graduated, area of their practice, type of practice, or availability of the therapist. This contrasts with a much greater incidence of emotional symptomatology in their practices. This discrepancy may reflect the problems inherent in the use of a loose definition of emotional illness. It may be that to many physicians the degree of incapacitation necessitates psychiatric referral, and that there may be an inclination toward treatment by the primary physician of the less disabling problems. One third of the respondents do tend to treat emotional illnesses themselves; but the degree and type of problem, as well as the methods used, were not established. That physicians have little time to totally evaluate and refer every patient with emotional problems may be another important factor.[1, 2] In addition, many physicians may refer patients to psychologists or community mental health centers which have no psychiatrists; thus they may be referring more than this survey measured. Schwab[3] suggests that fear of the patient's adverse reactions toward a psychiatrist may interfere with the referral process. In addition, he proposes that the mystery of psychotherapy may deter the process. However, most of our respondents felt the intervention of a psychotherapist was useful and relevant.

It is less likely that this discrepancy between referral rate and incidence of emotional illness reflects a negative attitude toward psychiatry, since the predominant mood reflected a greater need for psychiatrists, as well as a consensus of good experiences with them. However, the assumption must be made that the segment of the sample of physicians who failed to respond may have different attitudes. No followup letters were mailed to them.

A significant tendency exists for physicians graduating from medical school before 1950 to restrict psychotherapy to physicians only, whereas those graduating after 1950 indicate psychotherapy may involve non-physicians as well as physicians. This may reflect less rigid attitudes toward psychiatry as a medical specialty, differences in the understanding of emotional illness, more recent emphasis during medical school training on involvement of paramedical professionals, or the younger physicians' interests in the psycho-social aspects of the patient. Grinker[4] supports this last explanation. Cleghorn[5] explains the situation as one of the more recent trends toward the synthesis of humanism and science in psychiatry. Regardless of the year of graduation from medical school, most of the respondents still referred few of their patients with emotional illness to psychiatrists. This is the most consistent finding from the survey, and its meaning remains speculative. Further investigations with pilot studies and followup letters are needed in the future.

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PULMONARY THROMBOEMBOLISM SYMPOSIUM

An institute on "The Prevention, Diagnosis, and Treatment of Pulmonary Thromboembolism" will be held on October 27, 1973 at the V.A. Hospital, 2200 Gage Blvd., Topeka, Dr. Howard N. Ward announces.

The faculty will consist of Arthur A. Sasahara, M.D., Harvard Medical School; Jack Hirsh, M.D., McMaster University Medical School, Hamilton, Ontario; and Kazi Mobin-Uddin, M.D., University of Kentucky School of Medicine.

If sufficient interest is indicated, a program for wives will also be available.

For reservations or information, please write: Dr. Ted Warren, Stormont-Vail Hospital, Topeka 66606.

Pericardial Cyst

500-Gram, 15 x 8 x 8 Cm.

JOSE M. VERA-ROMAN, M.D.* *and*
DON R. MILLER, M.D.,† *Kansas City, Kansas*

PERICARDIAL cysts are relatively common lesions and usually do not present problems in diagnosis or treatment. The cause of the disease is considered to be congenital.

A pericardial cyst recently treated had the unusual characteristic of large size, and posed questions as to the diagnosis and cause believed worthy of a case report.

Clinical Summary

A 69-year-old white male presented with easy fatigability and a large right paracardiac radiopaque mass. This was in ill-defined continuity with the cardiac silhouette, pulseless, and not attached to the corresponding hemidiaphragm *(Figure 1)*. Laminograms showed that the mass was definitely separated from the postero-inferior margin of the heart, but strongly suggested a primary tumor of mediastinal origin.

Approximately one year before this hospital admission, the patient sustained fractures of his fourth and fifth right ribs in the lateral thoracic segment. Apparently he recovered satisfactorily. There were no other pertinent clinical findings. Attempted needle aspiration by his family physician was unsuccessful. Preoperative diagnosis considered, in addition to pericardial cyst, was bronchogenic carcinoma, cardiac tumor or aneurysm, and bronchogenic cyst.

At operation, the mass was a cyst attached to the pericardium by a broad pedicle and some fibrous adhesions. There were also fibrous adhesions to the middle lobe; the surgical excision and recovery were uneventful.

Anatomical Pathology

The cyst weighed 500 gm and measured 15 x 8 x 8 cm *(Figure 2)*. It was translucent, ovoid with some lobulations, and contained clear-amber fluid. The inner aspect was smooth and glistening with small trabeculated areas. Microscopically, the cyst wall consisted of a fibrous core lined internally with a pseudostratified mesothelium and covered externally with vascularized fibroadipose tissue with scattered small foci of lymphocytes *(Figure 3)*.

Comment

Pericardial cysts do not generally present problems in diagnosis or management. Wychulis *et al.*[1] have recently reported a large series. Loehr[2] proposed the following classifications: (1) Congenital (true cysts)—coelomic, lymphangiomatous, bronchial or teratomatous; (2) Acquired, secondary to hematoma, neoplasm, or to parasitic disease; (3) Pseudocysts—pericardial diverticulae and encapsulated pericardial exudate. The coelomic variety is the most commonly found and the cyst is usually located in the right anterior cardiophrenic angle. Lambert[3] related their

An unusually large pericardial cyst, which presented diagnostic problems, was surgically excised. A history of chest trauma might have had etiologic significance.

Figure 1. Posterior-anterior chest film showing the large mass in the lower lung field continuous with the mediastinum.

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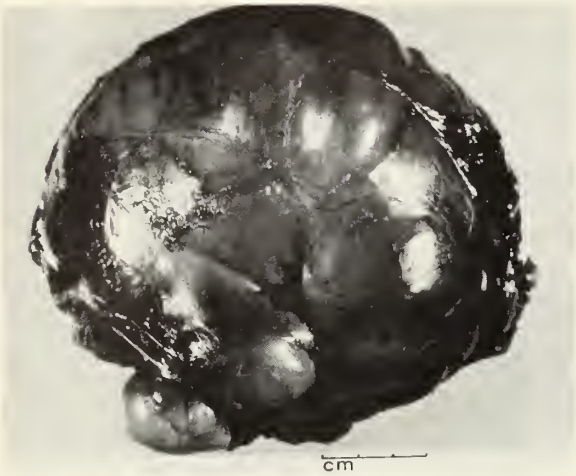

Figure 2. Gross photograph of the cyst which measured 15 x 8 x 8 cm.

Figure 3. Microscopic section of cyst wall.

origin to a failure of the pericardial mesenchymal lacunae to unite with the pericardial coelom.

The present case presented preoperative diagnostic difficulties because of the large size and contour of the mass. However, at operation and pathologically it had characteristics of a coelomic-type cyst. The possibility of its being related to traumatic origin is of interest. The traumatic episode may have resulted in exudation of abundant fluid, hence the large size. The vascularized fibroadipose tissue on the external aspect was compatible with healed inflammatory changes.

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The Fall Meeting

of the

KMS HOUSE OF DELEGATES

Sunday, November 18, 1973—10:00 A.M.

University Ramada Inn, Manhattan

One-day business meeting to consider items relating to legislation and others that cannot be delayed until May. It is hoped all component societies will be represented by their full complement of delegates.

All members of the Society are invited.

ALL RESOLUTIONS MUST BE SUBMITTED NOT LATER THAN NOVEMBER 1, 1973. THEY WILL BE REPRODUCED IN SUFFICIENT QUANTITY TO PASS OUT TO THE DELEGATES AT THE MEETING.

KUMC Trauma Conference

Multiple Stab Wounds of the Head, Neck, and Thorax

Edited by **ARLO S. HERMRECK, M.D.** *and*
FREDERICK W. RECKLING, M.D., *Kansas City, Kansas*

Dr. Haskey (Surgical Resident): The patient for presentation today is a 40-year-old white male who was transferred from the Lansing State Prison after being assaulted and stabbed multiple times. The patient related that he was attacked by a fellow prisoner with a self-constructed knife and stabbed in several areas of the body. The assault ended when the last stab wound to the head resulted in penetration and retention of the weapon in the skull.

Physical examination of the patient in the KUMC emergency room revealed the blood pressure to be 100/60 mm Hg, pulse 110 beats/minute, and respiration 20. There was a large metal object projecting from the left temporoparietal area. The patient was awake, alert, and able to converse with the interviewers. His extraocular eye movements were normal, and his pupils were equal and reactive to light. He could move all four extremities, and his sensation was intact except for the left upper extremity.

More specific examination revealed a stab wound of the left side of the neck just above the clavicle. In the left shoulder area near the apex of the axilla there was a massive hematoma, and a through-and-through lateral knife wound in the triceps area of his left upper extremity. In addition, he had a stab wound of the chest at the right 4th intercostal space along the anterior axillary line. There was a small amount of air escaping from this wound and also some subcutaneous emphysema was palpable over his right lateral chest area. His heart sounds were normal with no murmurs. Examination of the chest revealed good bilateral breath sounds. There was no evidence of trauma to the abdomen or lower extremities. The left hand was cold and pale, and there were no ulnar or radial pulses palpable. There was also lack of sensation in the dorsal webb space of the left hand. In addition to this, neurological examination revealed loss of extension at the wrist and also some weakness of extension at the elbow.

Dr. Reckling: Could you describe the knife?

Dr. Haskey: It apparently was made from an old drill bit that had been honed by equipment at the prison.

Dr. Reckling: Dr. Haskey, would you continue with the patient's course.

Dr. Haskey: The patient's condition was stable and the mild hypotension present upon admission responded well to fluid administration. A Foley catheter was inserted and good urine output was noted. Since the patient's cardiovascular status was quite stable, we consulted the neurosurgeons for further evaluation of the patient. Their recommendations were that the patient have an arteriogram of his cerebral vessels to delineate whether or not major intracranial vessels had been violated with this knife. In addition, consultation was obtained with general surgery at this time, and it was recommended that the patient have a left subclavian arteriogram at the time of his cerebral arteriogram to show exactly where the vascular supply of his arm had been interrupted. In addition to these x-rays, chest and skull films were obtained.

Dr. Reckling: Dr. Haskey, would you like to comment on these x-rays?

Dr. Haskey: First of all, we might look at the A-P and lateral view of the skull *(Figures 1, 2)*. As you

Figure 1. A-P view of the skull showing the intracranial position of the knife.

Figure 2. Lateral view of the skull. Note the bent tip of the knife.

Figure 3. A-P view of left cerebral arteriogram.

can see from these films, there were two or three inches of steel projecting intracranially. This instrument had entered the cranial vault on the left side at the temporoparietal area. From these x-rays you can see that the instrument projected into the parietal area and that the tip of the knife had actually been bent upon penetration of the skull. The chest x-ray reveals a small pneumothorax on this right side along with the minimal subcutaneous emphysema. In particular, we were interested about whether or not there was mediastinal air. None was noted on the initial chest x-ray. The next x-rays to be reviewed are the arteriograms of the extra- and intracranial vessels on the left side *(Figures 3, 4)*. As you can see, there is excellent filling of the major intracranial vessels with no apparent severance by this sharp object. X-rays of the left subclavian and axillary vessels revealed total occlusion of the left axillary artery in the mid portion of the axilla. One can see some collateral vessels in this area, but the more distal axillary artery does not fill completely *(Figure 5)*.

Dr. Reckling: What was done for the patient at this point, Dr. Haskey?

Dr. Haskey: Because of the small pneumothorax, the patient had a chest tube inserted in the right pleural cavity and only a small amount of blood and air was removed. There was no air leak. At this point the neurosurgeons took over the patient's care, and moved the patient to the operating room for removal of the knife from the cranial vault.

Dr. Reckling: Did they take any special precautions in removing the knife?

Dr. Haskey: Yes. Because the projectile was a centimeter or so away from the primary speech center, they were concerned that if a major debridement was done they would destroy the speech center. Therefore, they did a craniotomy with removal of the knife and a very limited debridement of the wound tract. No significant hemorrhage was noted, and the patient tolerated the procedure well.

Dr. Hermreck: Dr. Maxwell, would you care to comment on whether or not there is an increased chance of infection because of a limited debridement of the wound tract?

Dr. Maxwell: Yes, I think there is a greater chance of infection. However, the alternative is debridement of a very deep penetrating wound of the brain which would result in gross neurological deficits. This we were not willing to accept.

Figure 4. Lateral view of left cerebral arteriogram.

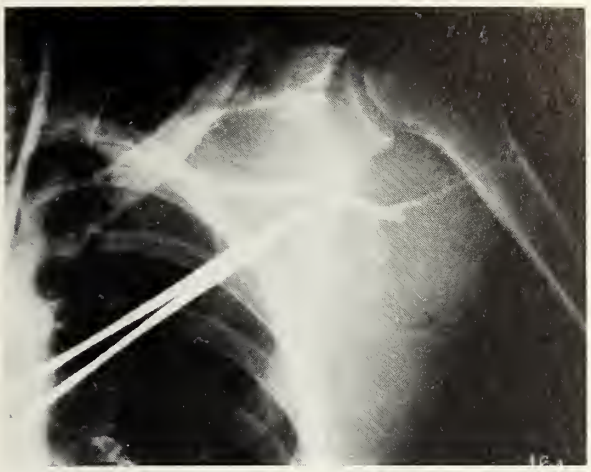

Figure 5. Arteriogram revealing total occlusion of the left axillary artery.

Dr. Reckling: Dr. Maxwell, what could have happened if this knife had been removed in the emergency room without turning a cranial flap?

Dr. Maxwell: First of all, this should never be done. The chances of a major intracranial vascular laceration with this type of injury are exceedingly high. Withdrawal of the knife can and does initiate hemorrhage with the formation of an intracranial clot and its sequelae.

Dr. Hermreck: Dr. Maxwell, what other precautions would you have taken had this instrument entered the lateral ventricle?

Dr. Maxwell: None.

Dr. Reckling: Dr. Haskey, what then took place with the patient?

Dr. Haskey: Following successful removal of the knife from the cranial vault, the case was then turned over to the general surgeons for the vascular repair of the left axillary artery and exploration of the neck.

Dr. Reckling: Dr. Hermreck, would you care to comment on what took place with the remainder of the operation?

Dr. Hermreck: As was mentioned previously, the patient had a stab wound in his left supraclavicular region, left axilla with a massive hematoma, and also a through-and-through stab wound of his left triceps region. Initially, we made a supraclavicular incision parallel to his clavicle on the left side and explored the neck. The stab wound in this area had penetrated into the anterior mediastinum and this was carefully explored. No definite injuries were noted to the anterior mediastinal structures. Prior to leaving this area, it was filled with water and forceful ventilation was carried out, and no evidence of an air leak was noted. Following this, the scalenus anterior muscle was divided and control of the left subclavian artery was obtained. Next, an incision paralleling the fibers of the pectoralis major muscle was performed. The axillary artery just distal to the insertion of the pectoralis minor muscle was encountered and found to be completely severed with the proximal and distal ends thrombosed. The left subclavian artery was clamped, the severed ends of the axillary artery mobilized, and the clots removed. A minimal debridement was carried out and an end-to-end anastomosis performed using 6-0 Tevdek suture. Exploration of the brachial plexus in the area did not reveal any injury. Upon removing the clamp from the subclavian artery, good flow was noted through the axillary artery and an excellent pulse was detected in the brachial artery. These wounds were closed primarily leaving a Penrose drain in the supraclavicular area and a Hemovac drain in the axillary wound. The patient tolerated these procedures well and was returned to the surgical intensive care unit, where he remained stable and was conscious for the remainder of the evening.

Dr. Reckling: Dr. Hermreck, do you want to discuss some of the technical aspects of arterial repair?

Dr. Hermreck: This was a low velocity type injury to the arterial system and, fortunately, one doesn't have to debride much tissue. Adequate mobilization of the transected ends of the vessel allows one to get the vessels approximated without tension and do a primary anastomosis. However, if this had been a high velocity vascular injury such as occurs with a gunshot wound, then it would be mandatory that one debride one or two centimeters of vessel on either side of the injury in order to get away from intimal fractures and the thrombosis problem that is so common after high velocity missile wounds. If this had been a high velocity missile wound, we, of course, would have harvested a segment of saphenous vein, reversed it, and carried out a grafting procedure. I would have been very reluctant to use a synthetic prosthesis in this area because of possible infection due to the dirty knife.

Another important principle in dealing with a vascular injury such as this, is to get proximal and distal control prior to exploring the severed vessel. We approached the vessel from the retroclavicular area, divided the anterior scalenus muscle, avoiding injury to his phrenic nerve, and obtained proximal control of the subclavian artery. In addition, the axillary artery was isolated distally in the upper arm to obtain control of the distal vessel.

Dr. Reckling: Did the neurological deficit in his left upper extremity disappear after vascular repair?

Dr. Hermreck: No, he had a persistent wrist drop, signifying an injury to the radial nerve.

Dr. Reckling: Dr. Haskey, would you continue with the patient's course?

Dr. Haskey: The patient remained stable throughout the night and was extubated. Upon extubation,

however, he was noted to have a small air leak through the left chest tube. At approximately 8:00 AM the following morning, the air leak stopped and the patient developed severe respiratory distress.

Dr. Reckling: Tell us more about this respiratory distress.

Dr. Haskey: The patient became severely cyanotic, was reintubated and placed on a MA-1 ventilator. He then developed massive subcutaneous emphysema on the right side extending into the neck, face, and scrotum *(Figure 6)*.

Dr. Hermreck: Was his chest tube functioning?

Dr. Haskey: No. I think this was what precipitated the problem. The chest tube stopped functioning, and the patient developed a massive air leak from an unknown source. His PaO_2 was about 40 mm Hg and his $PaCO_2$ was about 70 mm Hg. The measured air leak on the ventilator was approximately 500 cc per breath. The chest tube was removed and another inserted with some improvement.

Dr. Reckling: Was there a clot blocking the chest tube?

Dr. Haskey: Yes.

Dr. Reckling: Dr. Pierce, you were asked to see this patient. Would you comment on his respiratory problem?

Dr. Pierce: This patient posed a number of interesting respiratory problems. Upon admission, he was noted to have a small pneumothorax on the right side with subcutaneous air, and a chest tube was inserted. He had a stab wound over the right chest, and it was a reasonable assumption to conclude that the air leak in the right chest was due to a puncture wound of his right lung. He developed severe respiratory distress the next morning and at this point, as you have already heard, the chest tube was found to be obstructed.

This brings out an important point. A patient with a pneumothorax on positive pressure ventilation must have a functioning chest tube in place. It is a particularly dangerous situation in the operating room to have a patient anesthetized, under sterile drapes, who develops a pneumothorax with respiratory distress. The diagnosis of this condition can be difficult. So, whenever one makes the diagnosis preoperatively of a pneumothorax, even if it appears small, a chest tube should be placed as a safety feature. This man's safety feature quit functioning due to the obstruction, and he developed a tension pneumothorax with escape of some of the air into the subcutaneous tissues. It is difficult to quantitate volume leaks just by amounts of subcutaneous emphysema. Even a small leak over a long time will give marked subcutaneous emphysema. With a patient on the ventilator, however, one can measure precisely the volume of air leak by subtracting the expired from the inspired volume. In this case, there

Figure 6. A-P of chest taken at the time of severe respiratory distress.

was a discrepancy of 400 to 500 cc's.

Because of this large air leak, we were concerned about a tracheal laceration. There was a stab wound in the left neck and a stab wound in the right chest, either of which could have penetrated the trachea. To evaluate this, a flexible fiberoptic bronchoscope was passed through the indwelling endotracheal tube and the distal trachea, carina, right and left main stem bronchus inspected. There was no blood or signs of major airway penetration. The upper trachea was inspected by deflating and withdrawing the endotracheal tube. Again, no leak or any evidence of injury was noted. The patient was observed for another two hours and the endotracheal tube changed and repositioned with no improvement in his respiratory status. The air leak progressively increased to about 1000 cc's/breath forcing surgical intervention. He was taken to the operating room and a right thoracotomy performed. A 5-mm laceration of the membranous portion of his trachea was found just below the thoracic inlet. This was closed with simple interrupted 4-0 Tevdek sutures and buttressed with a flap of pleura over the closure. The repair was tested with 40 to 50 cm of water pressure intratracheally with no evidence of leak.

It is interesting to speculate as to how this wound occurred. Was it the stab wound to the right chest, or was it the neck wound? Also, why couldn't we diagnose it with the optic bronchoscope. Possibly, the endotracheal tube had obscured it enough so that visualization was impossible with the bronchoscope. One serious problem with the fiberoptic bronchoscope is depth perception. Perhaps this is why this injury was missed.

Dr. Reckling: Did you find a lung injury at thoracotomy?

Dr. Pierce: He did have a small puncture wound of his right upper lobe that had sealed off. It wasn't leaking at the time of thoracotomy, and it was directly underlying his anterior chest stab wound.

Dr. Hermreck: Maybe we ought to have Dr. Pierce tell us about how one differentiates between a tracheal injury or a major bronchial injury versus a peripheral laceration of the lung.

Dr. Pierce: This can be a difficult differential. The major thing we look for with a tracheal injury or a major bronchial injury is the volume of leak. Peripheral injuries are more likely to give a small volume leak, and subcutaneous emphysema is more concentrated at the local injury site. The diagnosis can be best established by bronchoscopy. Usually, the combination of massive leak and bronchoscopic findings establish the diagnosis.

Dr. Hermreck: Dr. Miller, how would you determine whether or not an esophageal injury was present?

Dr. Miller: This is a very critical problem and certainly must be dealt with. The patient with an esophageal injury can also develop subcutaneous emphysema or mediastinal emphysema. Usually, a patient with an esophageal leak has mediastinal air. If not, the only good way to rule out this type of injury is to have the patient swallow some contrast media and look for extravasation by x-ray. This is an injury that is frequently overlooked, and the consequences of it can be very devastating if a leak occurs and goes untreated for 24 or 48 hours.

Dr. Reckling: Dr. Haskey, how has the patient done since the thoracotomy?

Dr. Haskey: Very well. No further air leaks occurred, and the patient was extubated after 24 hours with good blood gases and without mechanical difficulty. All wounds healed primarily, and excellent radial and ulnar pulses are present in the left arm. The only neurological deficit is the radial nerve injury in the left arm.

Dr. Reckling: Thank you all for participating in this conference.

Addendum: Dr. Maxwell explored this patient's radial nerve in the upper arm and found a complete severance by the stab wound in the triceps region. A primary anastomosis was performed. The patient has made an uneventful recovery from his injuries, although it is too early to evaluate his radial nerve recovery.

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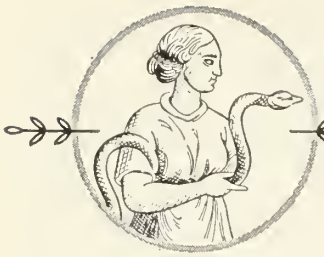
YOUTH BLOOD CORPS FORMED

Anticipating the federal prohibition of purchased blood by 1975, the Topeka Blood Bank, Inc. has turned to a logical source of interest and manpower in preparing to go on an all-volunteer basis. During the past summer, a group of 25 high school students, under the guidance of Antonio Huaman, M.D., Topeka pathologist and president of the Board of the Topeka Blood Bank, has developed the Topeka Youth Blood Corps. This group of young people has assumed the obligation to conduct a door-to-door campaign to set up a registry of volunteer blood donors. Under the plan, the volunteers will agree to donate at least one pint of blood annually as required by demands on the bank. The Youth Blood Corps will be a continuing organization, assuring that community involvement will be sustained and an adequate supply of donors will be available to meet the annual 6,000 pint requirement of the Topeka bank.

Beside making available an expanded registry for general blood needs, it is expected that the efforts of the corps will increase the number of persons on the special roster of rarer blood groups which has been maintained by the blood bank. The Topeka Blood Bank foresees that the plan, in addition to directing the interest and enthusiasm of young people in this age group into a community effort of prime importance, will be a valuable agency in community education regarding blood banking and utilization, as well as preparing young people for active participation in the program after they pass the minimum donor age of 18.



Medical HISTORY

Isaac Arthur Abt, 1867-1955

RONALD D. GREENWOOD, M.D.,* ***Boston, Massachusetts***

THE STORY of Isaac Arthur Abt, M.D., is the story of pediatrics in the United States. He is undoubtedly Mr. Pediatrics of Chicago, and vies with Abraham Jacobi (1830-1919) in importance nationwide (Jacobi is the "Father of American Pediatrics," for he held the first chair of pediatrics in the United States, and organized various pediatric societies and journals). Arey states of Abt, "Perhaps no other American pediatrician was as well known and highly esteemed throughout the world."

Isaac Abt, the first of twins, was born on December 18, 1867, in Wilmington, Illinois. His first association with medicine was with a disease which one day he would help to conquer. The disease was diphtheria. He tells the story in *Baby Doctor:*

The doctor gave the usual local treatment, but the child's condition did not improve. Early one afternoon my grandmother and father set off to see if they could be of service to the child. My brothers and I watched them go. They had not returned by our bedtime, and when we went down to breakfast in the morning only mother was in the kitchen. She was very quiet. In fact, all of us were subdued and scarcely needed to be told to leave the room when grandmother and father finally came home. We were not supposed to hear the details of their experiences, but every one was too absorbed to notice a little boy lingering just outside the door. I saw my father shake his head in answer to mother's unspoken question.

"It was terrible," he said. "They tried everything, but the child couldn't get his breath. At last the doctor had to make an opening in the windpipe and put in a tube to keep the boy from suffocating. He was so sick you wouldn't have thought he could resist, but his struggles were frightful."

Mother spoke huskily, "And didn't that—?"

"It was no use."

I slipped away to tell my twin brother what I had heard. We whispered together very solemnly and put our hands to our throats, imagining the agony of choking, sensing the tragedy of a child's death without understanding it.

When Isaac was eight years old, his family moved to Chicago. That was in 1875. Yet, his exposure to medicine and horrible diseases had not ended. A cousin had died of scarlet fever and his sister, four years younger then he, had a severe type of the disease. His youngest sister died of diphtheria at the age of six.

At the age of 12 or 13, he began to work after school in a drugstore for a druggist who would not only give advice but would pull teeth, lance boils, or remove ocular foreign bodies. His duties here ranged from cleaning bottles to grinding herbs and roots.

In 1886, Dr. Abt began his college career at Johns Hopkins University. He never regretted his choice of a university, for the faculty included such men as Ira Remsen, Henry Newell Martin, Willian Keith Brooks, and William Henry Welch. During winter and spring vacations, he met his brother, who was attending Yale, in New York and engaged in debates, especially on immortality. His brother, who was deeply religious, tried to convert Isaac from the materialistic view of life, but he stubbornly resisted. Whatever his family thought about his religious views, they supported Isaac in his medical aspirations wholeheartedly.

Summer vacations were spent in Chicago seeing clinical applications, attending lectures of Christian Fenger and Nicholas Senn, acting as assistant to Dr. Edmund Andrews, Sr., and working as a clinical clerk at Mercy Hospital.

While in Baltimore for his third year of undergraduate work, Abt had to think about choosing a school for medical training. Hopkins had no medical department until 1893, so Abt had to consider another

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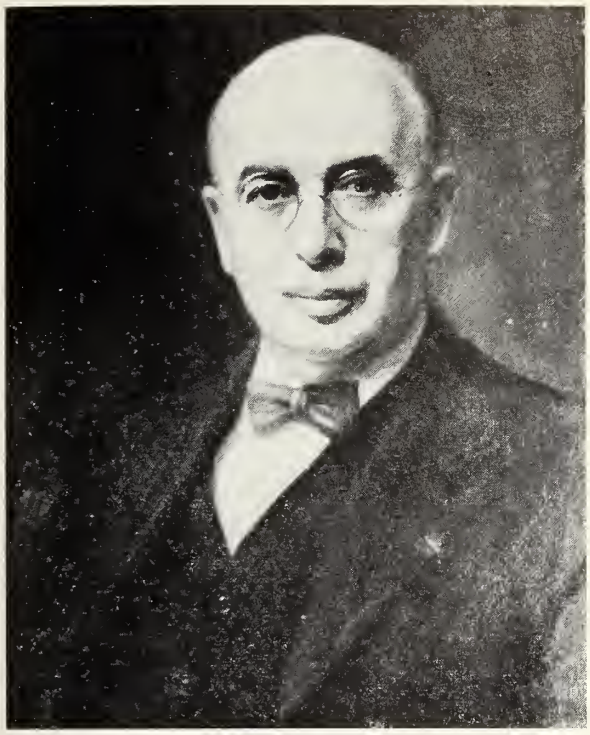
The opinions expressed herein are those of the author and not of the U. S. Department of the Army.



Isaac A. Abt

school. Dr. Welch assured him that none was too good, so Abt decided to return to Chicago Medical College, since it was "far in advance of most of the older institutions" even though it had many shortcomings.

During his second year at medical school, Abt, Adolph Gehrman, Dan Eisendrath, and Dr. Bayard Holmes began the study of bacteriology in a closet under the amphitheatre. This was only seven years after Koch published his investigations on the tubercle bacillus.

Abt spent his vacations reading medicine in the offices of Drs. W. K. Jacques and E. V. McDonald. He graduated in 1891; included among his classmates were DeLee, Preble, Edwards, Walls, Eisendrath, and Schroeder.

That year, he began his internship at Michael Reese Hospital, which at that time was only a 100-bed hospital. His experiences there were a mixture of very hard work and scientific investigation. Although he was "busy day and night," he published some studies in the *Chicago Medical Recorder,* "A Report of Six Cases of Suppurative and Tuberculous Diseases of the Brain and Meninges." He notes:

> More and more, however, my interest was turning to the diseases of children. Here there was a gap in medical knowledge. I had seen it during my student days, when it was apparent that, for the most part, diagnosis of children's diseases was based on superficial knowledge, and physicians were not getting the best results. My contacts with children in the hospital and dispensary and ward heightened my interest in pediatrics but did not contribute significantly to my knowledge of the subject. I read the works of the various authorities; Abraham Jacobi, Edward Henoch, J. Lewis Smith, Herman Widenhofer, and they only served to make me more aware that I had a vacuum which I knew I must fill.

From this date on, pediatrics would have one of its most ardent scholars and clinicians. When Abt discussed his future with older doctors, they either smiled patronizingly or tried to discourage him. "The diseases of children are the specialty of the general practitioner," they told him, "there is no future in pediatrics." It was true that there were only four or five outstanding pediatricians in the United States, but this only caused Dr. Abt to be further convinced that he should become a baby doctor and that he should pursue his studies in Europe. He finished his internship in September 1892, and left for Vienna.

In Vienna, Dr. Abt studied with Professor Herman Widerhofer, head of the department of pediatrics at the University of The St. Annen Children's Hospital, where Abt concluded that the Viennese professors were far in advance of most of their American colleagues.

From Vienna he went to Berlin, where he worked with Dr. Adolph Baginsky at the "best equipped and most modern hospital in the world." Here he again met up with diphtheria. The treatment for this disease was ice packs around the necks of the children. "In those days, many children who sat up in bed fell over lifeless from the effect of the diphtheria toxin on the heart." That was such a common occurrence that parents were warned not to let their children sit up.

From Berlin, Dr. Abt traveled to London where he attended clinics at the Children's Hospital in Great Ormond Street, and the hospital for epileptic and nervous diseases in Russell Square, but he got very little to advance his knowledge of pediatrics.

After 18 months of European study, Dr. Abt returned to Chicago to begin his practice of pediatrics. He was now 27 years of age, bearded, and most eager to begin his practice; he chose an office, a five-room flat above a drugstore at 35th Street and Indiana Avenue. The only children brought to him, however, were the sons and daughters of relatives, although he did have other patients. The first of these had the chief complaint of, "It's his tail, sir. They've cut it off, don't you see, but it's not right. We couldn't enter him in the show looking like this, could we, sir? And he's a good dog." Dr. Abt proceeded to do a flap operation removing several disks of bone with a carpenter's chisel. His other patients included a dog run over by an Indiana Avenue horse car, who made a good recovery, and a sick canary, whose outcome

was not so good. This case he followed with an autopsy. These were dark days indeed for the child specialist.

Soon after his return to Chicago, he was appointed assistant in pediatrics and instructor in physiology and histology at Northwestern University Medical School, and was put to work to create the first physiological laboratory there. He began this task with great enthusiasm and spent much time going over texts, making tissue preparations, and physiological demonstrations. In addition, he held pediatric clinics at the medical school, Michael Reese Hospital, and the West Side Dispensary. A little later, he was appointed professor of diseases of children at the Women's Medical College of Northwestern University, and attending physician at the Old Peoples' Home and the Jewish Orphan Asylum, and soon succeeded his former instructor, Dr. Hatfield, as pediatrician at the Chicago Orphan Asylum.

During these early days of practice, Dr. Abt was again involved with the disease with which he had his earliest exposure to medicine, diphtheria. And this time, unlike all the ice pack treatments in Europe, Dr. Abt would conquer the disease. The year was 1895. One night, a general practitioner called Dr. Abt and informed him that a patient of his would probably not recover from an attack of virulent diphtheria, and asked Dr. Abt to try vonBehring's new antitoxin. Abt managed to obtain some and rushed to the child.

> I gave the antitoxin and we waited. To my knowledge, this was the first time the serum had been administered in Chicago. Neither of us had ever seen its effect. The child lay still; his skin was blue, his pulse very weak; but he lived through the night.
>
> In the morning, his temperature was lower, he was breathing regularly, and color had returned to his lips. Twenty-four hours later, all immediate danger was past; and in the days that followed, we saw him make a quick and uneventful recovery.

In those days, Dr. Abt performed minor surgical operations, treated injuries, reduced simple fractures, delivered babies, removed many tonsils and adenoids, and opened many retropharyngeal abscesses.

He held public office twice in those early days; first, as district county physician and then as inspector on the staff of the Chicago Health Department. In this latter post, he had many interesting and some dangerous experiences.

Part of his work as health inspector was to visit homes where there was communicable disease; and this involved a constant battle with ignorance, suspicious resentment, and religious taboos. Most rebellious of all were those whose religious beliefs were opposed to medicine. Foremost among these were those who followed a man named Dowie, who was an evangelist and faith healer in Chicago at that time. Abt avoided boiling water, razors, and other incidents by promptly seeking the help of the police.

By 1896, the young Abt became instructor at Northwestern and held that position for six years. At that time, Marcus P. Hatfield, the first real pediatrician at Northwestern resigned, and Dr. Abt felt his appointment as Hatfield's successor was eminent, and deservedly so. However, the dean at that time, Nathan Smith Davis, Jr., appointed Francis X. Walls as the second professor of pediatrics. Dr. Walls was an old friend of Abt; they were roommates at school. Walls was a man of ability, but he had no particular training in pediatrics and had previously engaged in teaching pathology, surgery, and internal medicine. Abt inquired as to why he had been passed over. Davis did not explain why, but merely told Abt that he could go just as far in his private practice as he could as a professor. Dr. Archibald Church, however, gave Abt a reason. Abt had been charged with favoring certain students who belonged to his medical fraternity. This was most upsetting for Abt, who had no idea of his student's affiliations and was not active in his fraternity. Abt felt this was a "little short of tragedy." For Northwestern Medical School, it was not that: it was a tragedy. And, had it not been for an ironic turn of events a few years later, the leadership of pediatrics in Chicago and the country would most likely have been elsewhere, and the rich heritage that Northwestern enjoys would have been thrown away by a few shortsighted men. Abt resigned and was appointed extramural professor of pediatrics at Rush Medical College.

In 1902, Abt became editor of the *Yearbook of Pediatrics*, a position he held until 1943. This book covered the yearly developments in pediatrics, and Abt reviewed thousands of articles. His private office was soon moved from Wentworth Avenue to a downtown office in the Reliance Building. In this period of his life, Dr. Abt fought typhoid fever in the finest houses on Michigan Avenue and Lakeshore, as well as in the slums on Maxwell Street. He reported on his work to both the Chicago Medical Society and then the Illinois State Medical Society in 1902. It was that period in which the world was shocked at the Iroquois Theatre fire in Chicago; approximately 600 persons died in the holocaust.

In 1903, Abt was admitted to the American Pediatric Society. At that time, membership was strictly limited and consisted of 50 men. In that same year, Abt began his fight against unsanitary milk, by his participation (as chairman) in the Milk Commission. Most hospitals even used inferior milk. The goal was to furnish needy children with clean pasteurized milk at nominal cost. Attempts at any legislation failed time and again, because the small milk dealers op-

posed the venture for they felt they could not pay the price of cleanliness. A state law went so far as to invalidate the Chicago milk ordinance, and the city council failed to accept a new bill. There was a mass protest, and Abt, with Miss Jane Addams of Hull House, Dr. Frank Billings, and Professor Paul Heinemann, bacteriologist at the University of Chicago, spoke. Abt's speech evoked mass antagonism among the people present, and he and all reformers were denounced. Alderman Hay of Chicago led the opposition forces and their denouncement. Abt replied that he had come to the meeting to represent the people and protect the lives of their babies. But, then as now, the price was too high to pay for the proper action. Shouts of "Put him out" rose from the crowd. Undaunted, Abt saw that a milk ordinance was soon passed, and there was a marked reduction in the incidence of tuberculosis, septic sore throat, and typhoid fever.

In 1909, Abt was given the opportunity to return to Northwestern. Dr. Arthur Edwards had succeeded Dr. Nathan Smith Davis, Jr., as dean of the medical school. Abt would be professor of pediatrics and chairman of the department. Abt went to Billings at Rush, to explain this opportunity and his decision. Billings tried to dissuade Abt.

"You Blamed Fool!" Dr. Billings thundered. "Why should you want to go there? Northwestern."

Abt tried to explain that he felt gratified to have the position he once was denied offered to him and that the new hospital at Michael Reese he was working on was near Northwestern.

Billings, after a long series of tries, gave up his fight and told Abt, "Do what you want, but I can promise you one thing: you'll never be happy at that place!"

Abt, who had a high regard for Billings, realized in later years that this was one of the few mistakes Billings ever made, for he was very happy at Northwestern. He was chairman of pediatrics there from 1909-1939. During this period, Abt made many achievements—one of these was in changes in and building of children's hospitals.

Since his first hospital affiliation, Abt never ceased to have problems with hospital administrators. As an attending pediatrician in 1900 at Cook County Hospital, he disliked the facilities. The pediatrics section consisted of two levels—a main floor for older children and balcony for infants. There were no means of separating the infectious ones, and in general, the place was dark and gloomy. But, more than all that, most of the babies were undernourished. They got dirty milk and were refused many other items, for instance, cod liver oil and oranges. When Abt demanded oranges from the warden, the reply was, "Cook County can't buy oranges for babies."

As Abt said, "Argument was futile. Human life was cheap, and oranges cost money. If a doctor felt that his patients had to have citrus fruits, he could only go out and buy them himself."

This ward for children was in poor condition and the administration made plans for a new children's pavilion. However, as Apt puts it, "They were good plans, but the best laid schemes of mice and men are apt to suffer severe mutilation when they are put through a political machine. By the time the various boards and officials finished cutting down here and substituting there, we had a badly arranged, poorly constructed building. The cheapest materials had been used and many necessities omitted."

But, these problems existed in children's departments everywhere. The periodic outbreak of infectious diseases and the inability of administrators to recognize that the child needed more and a different kind of care were at the root of the problem.

At Michael Reese Hospital, the conditions were better because that hospital was rebuilt in 1907. However, again there were problems. The superintendents' qualifications were merely political connections, and blunders of large magnitude were made.

In 1909, before the move back to Northwestern, Mr. Edward Morris requested Dr. Abt to build a children's hospital for which Morris would supply the funds. Abt's first thought was to have it associated with Michael Reese Hospital, for it would enjoy certain economic advantages. Morris would rather have had a separate institution, and afterwards Abt regretted his linking the hospital with Michael Reese.

The hospital was to be the Sarah Morris Hospital, and would incorporate the best features of the best hospitals in the world. Abt then set off for Europe to see the institutions of London, Paris, Berlin, Munich, and Vienna.

The hospital was built and was a fine hospital. People came from all parts of the country to see it. Then, the problems began.

Michael Reese began to make demands, most of them basically ridiculous, and soon the endowment for the children's hospital was gone. The Sarah Morris was only an ornament to the Michael Reese, and was far from an equal partner. Administrators again plagued Dr. Abt. The nurses at the Morris were rotated throughout the hospital, so no one stayed in pediatrics to learn the special care necessary for children. For instance, food would be brought to the bedside and left as if an adult were going to eat. The child in many cases could not begin to feed himself, but there were no attempts to understand such problems. The children's building was allowed to deteriorate drastically. The reason was a superintendent of the hospital. Dr. Abt and his wife fought a valiant fight against the decay of this hospital in facilities, and the future decay in its attitude toward

children. Finally, in 1925, Dr. Abt resigned from Michael Reese. He then joined the staff at St. Luke's Hospital and stayed there until 1932. Then, he became an attending physician at Passavant Memorial Hospital and a consultant at Children's Memorial Hospital.

One of his dreams, the Isaac Arthur Abt Hospital, which would have occupied a desirable corner of Northwestern's McKinlock Campus, had advanced as far as the blueprint stage, but it never became a reality due to the 1929 depression.

Abt continued to be an excellent teacher, clinician, and researcher until his retirement. He was joined by his son, Arthur F., in his practice, and this allowed him much more versatility.

As a student, Dr. Abt was exceptional. In fact, throughout his professional career, once each month, a study club composed of 20-30 people met in his study to consider and discuss diseases. They assigned themselves topics and reported according to their specialties on the available information.

Some of the participants were Dr. Ross Y. Koskins, professor of physiology; Arthur Kendall, professor of bacteriology; James Simonds, professor of pathology; Leslie B. Arey, professor of anatomy; Chester Farmer, professor of biochemistry; Joseph Friedman, internist; Alfred Strauss, surgeon; D'Orsay Hecht, neurologist; Oscar Schultz, pathologist; Alexander Day, professor of bacteriology; Julius Hess, Abraham Levinson, Jesse Gerstley, Mark Jampolis, pediatricians. Visiting dignitaries were invited to the sessions or special meetings were called to coincide with their visits.

Residents in pediatrics were invited to these study group meetings. At one session, the discussion began about the new class of student nurses that had arrived at the hospital.

"Did you meet with the nurses?," Abt asked. "What did you talk to them about?"

Around the room the question passed, each young pediatrician telling all the sophisticated scientific topics discussed with the nurses. Finally it came back to Abt.

"Did you meet with the nurses?"

"Oh, yes, once," was the answer.

"What did you tell them?"

"Oh, I just showed them the proper way to hold a baby," Abt replied. The great pediatrician had outdistanced the young clinicians.

Arey remembers these "delightful affairs." After the discussion, Mrs. Abt would fix some European dinner and serve coffee.

As mentioned, these meetings were held in Abt's study. This was a huge room on the upper floor lined with books, overflowing into stacks in the hall. At one point, an architect was consulted, and the library was judged to be a danger to the home. It wasn't built to withstand such weight and was in danger of collapsing.

As a teacher, Dr. Abt was dedicated. He would prepare carefully for his lectures and for clinical work. He would call the students to the bedside and show the positive findings. He was very interested in their learning and experimented with his examinations and teaching methods.

As a researcher, Abt's work was clinical and encompassed infectious disease, asthma, gastroenterology, psychological problems, feeding and nutrition, heart disease, genitourinary disease, poisoning, blood disease, endocrine disease, covering the entire spectrum of pediatrics. His scientific publications number close to 165.

Among Dr. Abt's achievements are the following positions or offices and honors:

For 16 years, he was a member of the House of Delegates of the American Medical Association, and chairman of the section on the Diseases of Children in 1911.

He was president of the American Pediatric Society in 1926, Chicago Medical Society in 1927, the American Academy of Pediatrics in 1930, the Institute of Medicine of Chicago in 1932, the American Association of Teachers of the Diseases of Children, the Children's Hospital Society, the Chicago Pediatric Society in 1903-9, and the Central States Pediatric Society.

He was an honorary member of the German Pediatric Society and of the Minnesota Chapter of Alpha Omega Alpha.

He was a chevalier of the Legion of Honor of France in 1927; he received the Distinguished Service Award of the American Medical Association in 1948 and the Sc.D. degree from Northwestern University in 1931.

His writings include a number of books: A translation of Hecker's *Atlas of the Diseases of Children* (1907); *The Baby's Food* (1917); *Yearbook of Pediatrics* (1902-43); *A System of Pediatrics*, 8 volumes (1925); *Baby Doctor* (1944); a 24-page pamphlet for American Medical Association distribution, *Keep Your Baby Well This Summer* (not dated) (with Julius Hess); and the section on History in Brennemann's *Pediatrics*.

Of Dr. Abt's autobiography, Dr. Irving S. Cutter wrote:

> *Baby Doctor*, a rather intimate autobiography by Isaac Arthur Abt, is the saga of a great heart. Self-effacing and honest, the tale is told simply but with groping power. It should be read by every father and mother in the land, by every teacher, by every one who has anything to do with babies, children, or young people. The author's homely philosophy and his wise counsels are set forth in the form of fascinating anecdotes and experiences.

The System of Pediatrics was begun before World War I and was delayed greatly by that holocaust. Many articles were outdated and had to be rewritten; some authors died, and many European authors were unable to contribute. Thus, this monumental work was even more monumental than it seems at first inspection.

Abt developed incubators for premature babies long before the modern mechanical devices were conceived, and he designed a motor-driven breast pump. The story of the latter is worth telling. Abt had seen so many babies suffer from the effects of artificial feeding and so many saved by simply giving breast milk, that he had long been interested in some method of supplying breast milk. Abt says:

At the Sarah Morris, wet nurses expressed the milk by hand, chiefly because the various breast pumps then available were not satisfactory. I was convinced that a motor-driven pump would be of great assistance in emptying the breast completely, and could be absolutely aseptic. For that reason, I had been studying the problem for some time. In fact, I had braved the laughter of my colleagues by attending every local dairy show for years, and by visiting farms to study mechanical milking devices.

He continued to search for a good device; although he constructed one on several different occasions, the results were never satisfactory. Finally, he drafted Edward Lasker, who was in the milking machinery manufacturing business, to study the problem with him. They studied books on the subject, estimated the pressure exerted by a suckling baby, and made further trials. The product of all this was an intermittent pumping device which was powered by an electric motor. The technique was complete and aseptic, and gained uniform praise from pediatricians.

Abt trained scores of pediatricians. Among his famous pupils were Joseph Brennemann, Clifford Grulee, Julius Hess, Stanley Gibson, and Robert Black.

One of these students, Grulee, wrote of Abt:

For those who knew him, his character and knowledge will be set examples that few men could equal. He was a source of inspiration to younger men and gave much of his life in instructing them.

Asked to describe Dr. Abt, Arey replied:

He was a kind of short, roley-poley fellow, five-five in height, kind of chubby although he lost weight in his later years. He was gentle, soft-spoken and inspired the confidence of mothers in a gentle way.

He continues:

He had a round, balding head. His twinkling eyes and soft speech betokened friendliness which effect a cropped mustache over full lips did not lessen. A neat bow-tie was so habitual that he could scarcely be envisioned otherwise. He was somewhat shy with strangers and his manner was humble. Yet, with a gentle and restrained deportment, went innate dignity.

Abt had a gentle sense of humor. Some examples are worth while. Abt had done some experiments pertaining to feeding children goat's milk. He bought 50 goats, gave them to Michael Reese Hospital, and they lived on the hospital grounds. Abt continues the story:

A Swiss orderly not only milked the goats, but lavished care and affection upon them that a patient might have envied. The children got along well on the new diet, though not much better than on good cow's milk. But the nurses lost the properly detached, scientific attitude toward the experiment when the male goat took to chasing and butting them.

Personally, I had no reason to regret my undertaking until a colleague presented my two sons with a goat. The boys arranged for this treasure to share the garage with our Stanley Steamer, and once, when someone left the hood open, he ate a meal of asbestos insulation. Luckily, he found the rest of the car inedible, but later he lunched with evident relish on the only copy of a speech I had just written for presentation at an important medical meeting. I had nothing left except my original notes, and found little to console me in the demonstration that my paper was at least digestible.

Abt reached retirement age (65 years of age) in 1932, after he had been teaching for 38 years. The school asked him to stay on and, although he had certain misgivings, he did. He also continued to be active in the House of Delegates of the American Medical Association, and in other organizations. Since children were not dying right and left as they had been in previous years, and with his son able to take care of his practice, Abt felt he could relax more and indulge in a desire he had held for a long time —to own a farm. Abt enjoyed watching things grow —animals, and the plants of the outdoors. So, he went about this project in his usual studious way; he visited an agricultural college, a number of agricultural stations and model farms, and returned to apply scientific methods on his farm. He soon found his planted crops had as many diseases as did the children he treated. He still had to be a baby doctor occasionally. And, he seemed to revert to his first days of practice in caring for animals. For example, he treated a horse for sunstroke and performed an autopsy on a sheep. Dr. Abt could not stop being a healer of the sick regardless of who or even what was sick.

Then it was 1940, and Abt had to reconsider retirement. He pondered, as he had in 1932, the aphorism of Gracian, "Do not wait to be the sun in its setting. A maxim of the wise is to leave before they are left. Know when to make a triumph of your exit; for at times the sun herself when most bright

will retire behind a cloud that she may not be seen to sink, thus to leave us in doubt as to whether she has set or not."

"Do not wait until men turn their backs upon you that they bury you, still alive in your feelings, but dead in their estimation."

His work was a great source of satisfaction, but he decided this time that it was time to step down from his post. After he retired, he still continued practicing pediatrics and editing the *Yearbook of Pediatrics,* and he even found time for his autobiography.

In November 22, 1955, Dr. Abt died of a coronary thrombosis preceded by carcinoma of the tongue.

In 1887, Abt had married Lena Rosenberg, a nurse at Michael Reese. They had two boys, one of whom, Arthur F., became a pediatrician, associate professor at Northwestern University Medical School, and author of *Abt-Garrison Pediatrics* in 1965.

Dr. Abt was a kind and a great man. His ideals in pediatrics brought about many changes in the development and practice of that specialty in the United States. He was discouraged from entering pediatrics, mistreated by his dean in his early years, ostracized by part of the populace for fighting for decent milk for children, and he fought terrific odds in establishing and attempting to maintain high standards of child care in hospitals.

Acknowledgments

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Non-Operative Visualization

(Continued from page 366)

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The President's Message

A senator from South Dakota, in an open letter to the President of the United States, stated recently: "Our energy 'crisis,' I believe, is deliberately contrived by the major oil companies to achieve a number of objectives which they are seeking." It seems to me that this statement could easily be changed to read: "Our health 'crisis,' I believe, is deliberately contrived by a group of politicians and social planners to achieve the objectives which they are seeking."

When we look at the "crises" which have primarily to do with economics, including fuel and food, one can easily understand that we are a country full of crises. I am not convinced that we have a crisis in health care. I am convinced that the vast majority of patients in the United States, and particularly in Kansas, have access to quality health care at any time of the day or night, on any day of the year, whether it be a holiday or not. This is not true for governmental employees.

Suppose we go to a completely politically structured health care delivery system as promoted by various members of our Congress. Could this destroy the incentive for the physician to work long hours and take care of a large number of patients? It appears to me that most physicians are aggressive and are willing to put out the amount of time and energy that it takes to handle their practices, and that they work best when they are going full blast. We are not overpaid when these things are taken into consideration. As a matter of fact, we enter the direct patient care area at a time of life considerably past any other profession—we end up having a shorter life span and thereby fewer productive years because of our profession.

A restructuring of the health care delivery system has been done over a good number of years as a slow, steady, progressional type of an evolution, and those things which have been good about the restructuring have been adopted throughout our profession, and those things which have been bad have been discarded. We have many controls placed on us through the consumers, known as our patients, and we do deliver good health care. Some of the politicians would have us radically alter this system; however, the vast majority of politicians, I believe, at this point in time, are with us all the way. We really do not have to be ashamed of the things that we have done, for most of our changes have been made in the best interest of the patient.

Thomas F. Taylor

President

Benefit Performance

Just how much thought the public is giving to changes in health care delivery and financing is problematic, but our journalistic opinion-givers assure us that it is a matter of prime public concern amounting virtually to revolution. As a matter of fact, physicians as a group are not far ahead of the public in their attention to these matters, which is one reason the medical approach has been one of reaction rather than action and the feeling has been that the best offense is a stubborn defense. The physician, focused on patient care, assumed that his "father-knows-best" attitude was unassailable. The cold fact of the present public mood is that he will have to come down off his pedestal and work things out along with everyone else who has a stake in health service provision.

It is high time, then (if not too late), for the medical profession to get across some basic facts of health economics which seem to have escaped the public in its outcry against the rise in health costs. We are not talking about the complaint of the individual patient about his individual bill. That is a matter between himself and his physician and if he wants to grouse about it, the physician's office is the place to do it. The problems of health care cost which are stimulating the changes are on a different level and will require changes in philosophy by the medical consumer as well as the medical purveyor.

One of the prime factors accounting for the rising cost of medical care has seemingly been overlooked. The public has been so busy viewing the cost with alarm, it has failed to see what the outlay has bought. In short, it has been buying life. Life expectancy has increased significantly over the last two generations, the same period of time which has seen greatest rise in cost. This lengthening of life is noted with some satisfaction by all, but it apparently hasn't occurred to them that it costs something.

We have expressed before our conviction that, rather than deny or seek ineffectually to disclaim the cost of health care, we should face the public with the fact that health care *should* be one of its major expenditures. The health planners have presented their staggering figures and alpine graphs as testimony to the need for change. Aside from the fact that every other segment of social effort can show comparable figures and graphs during these expansive, war-torn, inflationary years, we think they are right but for the wrong reasons. We are going to have change, but not because of what medical care has cost or in order that it be cheaper—it won't be—but because changes are being wrought in every area of our economy and we have no choice but to adapt—hopefully even to help implement the new order.

The benefits of past health service are not readily apparent. They do not rise above a base level as material and tangible entities to be appreciated in relation to their cost. Since life and health are counted rights, medical progress and improvements have been absorbed into the social fabric and serve only to further the inviolability of the rights.

What does it mean that life expectancy has been prolonged? For one thing, it has contributed to increasing the gross national product (which is presumably some measure of our economic health) by increasing the labor force and at the same time the demand for goods and services. (This extension of life is the more notable when we take into account the number of young people lost to wars and traffic accidents.) As life has lengthened, more people are able to continue working longer and defer retirement. Or retirement, which was formerly only a brief interlude before death, is followed by another occupation with continued contribution to the economy. There is no way to calculate the cost that has gone into this increase in life expectancy but it seems not unreasonable, if we are going to cite astronomical sums being spent in the health market, that some social bookkeeping system should be devised to set off these against the benefits which have accrued from this primarily medical effort.

The increase in longevity is not, of course, pure blessing, but even in its less fortunate aspects has some economic impact. Many of those individuals who are living longer are only poorly or not at all

productive. In fact, many must have unusual service and care in order to survive. Their contribution to the economy is, at best, indirect in their requirement of special care facilities, additional personnel to provide this care, equipment, medication, and other therapies. Nevertheless, all of these things go into those health care figures which are cited as criticism of the medical function.

Another element in health care costs (or perhaps a different view of the same one) that needs to be stressed is the part public demand plays in their increase. Individually, the patient wants the best care regardless, at the moment of his illness, of the cost. Collectively this produces a formidable bill. It means that constant research must be pursued, much of it a financial loss. The training of medical personnel at all levels becomes longer and more expensive. New procedures and equipment must be produced, soon to become obsolete but essential at the moment if the "best" is to be provided. All this is not contrary to medical wishes—it is, in fact, the natural method of progress in the medical discipline where optimum care is the carrot-on-a-stick goal. But it is not based on the whims and pleasures of the physician. It is a system which persists because man is unwilling to settle for something if something better can be produced—and it always can be. In health care, there can be only one grade of service, the best currently available, but it is paid for with coinage whose exchange value is determined in a much different type of market. Medical service is subject to certain economies which the profession is seeking as diligently as the government or any other group. In brief, the public and the profession have the same goal in mind, but the public should have a greater awareness that it, in expecting optimum care, is as much a factor in promoting the burgeoning medical costs as any philosophical or functional effect of the profession.

The public must realize it has contributed to profound social changes which have figured in the current cost of medical care. Increased hospital utilization has occurred not just because the physician is able to provide the currently demanded type of medical care more efficiently and conveniently, but also because more patients have more insurance coverage either directly or as employment benefits. Furthermore, the use of hospitals, increased length of hospital stay, the increased demand for extended care and chronic care facilities are reflections of the altered function of the home. The communal family unit with the younger caring for the elder has been affected by the greater mobility of the populace, with the younger perhaps living no longer in the same community or the younger being occupied with outside employment, so there is no one at home to care

(Continued on page 392)

"THE KILLERS"

A major effort to inform the public on medical matters will be initiated on November 19, when the Public Broadcasting System (including KTWU, Channel 11, Topeka, and KPTS, Channel 8, Wichita) presents the first of five programs covering the five medical areas accounting for three-fourths of deaths. Each program will be 90 minutes in length, and the individual subjects include heart disease, inborn genetic defects, pulmonary disease, trauma, and cancer. Each program has been developed around the prevention, early detection, and treatment of the particular disease group with the aid of medical advisory boards composed of recognized authorities in each field.

More than 50 national health-oriented groups are cooperating in the presentation and local stations will promote local community programs in cooperation with the local chapters of such groups to emphasize and increase the value of the programs to the public. The Bristol-Myers Company is underwriting the effort.

The initial program, "Heart Disease," will be presented on November 19, and will range from congenital heart problems to age-induced conditions. The advisory board for this program includes Campbell Moses, M.D., former medical director of the American Heart Association; Theodore Cooper, M.D., director of the National Heart and Lung Institute; Samuel Fox, II, M.D., professor of medicine at George Washington University; and Irvine Page, M.D., editor of *Modern Medicine*.

The series will offer local physicians a unique opportunity to participate in a medically sound effort to interest and inform the laity regarding these significant medical subjects. The dates for subsequent programs are:

December 17—Inborn Genetic Defects
January 14—Pulmonary Disease
February 11—Trauma
March 11—Cancer

Woman's Auxiliary

Katie's Keys (With Apologies to the Kappa Kappa Gamma Quarterly)

"We have the keys to a better understanding of HEALTH. Let's use them!"

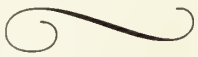
That's the theme for the Kansas Auxiliary this year with the admonition that we should use what we know and can learn together. Although we feel that some husbands are well aware of the pursuits and programs of the Auxiliary, many are not, and it is our intent to make our efforts more meaningful to all our doctors in Kansas!

An auxiliary is "a person or thing that gives aid of any kind; a helper." To be able to assist, we often need guidance ourselves so we can then carry out a plan or project that the doctors simply do not have time to undertake. So, first, we must educate ourselves through study of AMA programs and materials and community needs and by learning from and assisting other groups in our communities throughout the state. *Keys* symbolize our committees—bound together with the keychain of *membership*.

The first key should have a diploma and dollar sign attached—our one national fund-raising committee: the American Medical Association Education and Research Foundation (often shortened to AMA-ERF). Its sole purpose is to eliminate the financial barriers to medicine for all who are qualified and accepted by an approved training institution by (1) providing unrestricted grants to medical schools, and (2) enabling medical students, interns, and residents to obtain low-interest loans not otherwise available. Worthwhile? You bet! All kinds of gifts, watches, memorials, and Christmas cards raised over $20,000 for Kansas last year, with a national total of $965,-256.27, bringing the total since 1951 to nearly $10 million. Barton County, Kansas, won the county award for raising $104.08 per capita! The new national chairman, our own Edith Lessenden, is aiming for "a million for millions" and we have no doubt we shall make it!

We'll keep this short and sweet, but we hope that our keys will be able to attract your eye more than once to our column. Yes, we'll use the key to give *you* a better understanding of Auxiliary health projects and programs—all in *your* honor and for the community!

Katie Keys



LIBRARY SERVICES AVAILABLE

The Clendening Medical Library at the University of Kansas Medical Center now offers reference and interlibrary loan services to health care personnel of Kansas. The State Finance Council authorized expenditures beginning July 1, 1973, for a State Medical Library Program.

The service replaces the Kansas Regional Medical Program Library Network, for which federal funding ended June 30, 1973, after being in existence for five years. Continuance of the service after June 30, 1974, will depend on the provision of funds by the Kansas State Legislature.

The Clendening Medical Library State Services provides search services and document delivery to physicians, allied health personnel and medical libraries. Three additional people have been added to the Medical Center staff to provide these services throughout the state. The Clendening Library will answer requests with the help of a toll free telephone service (800-332-4193) and MEDLINE, a computerized reference list initiated by the National Library of Medicine.

Medical-Legal Page

Suit for Injuries Caused By Oral Contraceptive

A complaint based on injuries allegedly sustained as a result of using an oral contraceptive pill was sufficient to state a cause of action, a New York appellate court ruled. A section of the Uniform Commercial Code providing that a buyer must notify a seller within a reasonable period after discovery of a breach of warranty did not apply.

A woman began using Oracon, an oral contraceptive, according to her physician's instructions in the summer of 1966. Although a blood spot developed in her left eye, she continued taking the pills until the summer of 1968, when another physician told her to cease taking them.

The woman brought action against the drug manufacturer and two physicians to recover damages for personal injuries and medical expenses. The trial court granted a motion by the drug company to dismiss all causes of action against it.

On appeal, the court found that dismissal of the cause of action for breach of warranty because of failure to notify the drug company within a reasonable time was error. The court said that the question as to whether timely notice was given within the meaning of the Uniform Commercial Code did not apply in a case such as the present one.

The appellate court also reinstated the cause of action for medical expenses. The court said that since the physicians had not moved to dismiss, it was not correct to dismiss the cause as to them. The court found that the cause must also be reinstated as to the drug company because of the validation of the cause for breach of warranty.—*Fischer v. Mead Johnson Laboratories,* 341 N.Y.S.2d 257 (N.Y.Sup.Ct., App. Div., March 5, 1973)

Damages Awarded for Negligent Back Surgery

A patient who suffered injuries as a result of allegedly unnecessary and negligent back surgery was awarded $595,000 by a California jury.

The patient, a 56-year-old medical records librarian, consulted a physician because of severe back pain. The physician had her admitted to a hospital and placed in traction. He performed a myelogram on her fourth day in the hospital and a laminectomy at L-5, S-1 three days later.

As a result of the operation the patient suffered arachnoiditis, causing weakness of the left leg. She also had bladder and bowel dysfunction, loss of libido, and back pain.

In an action against the physician and the hospital, the patient contended that the surgery was unnecessary and that it had been negligently performed. She also alleged that the physician was negligent in failing to provide sufficient information for her to give an informed consent. She said that the postoperative care was negligent and that inducing her to submit to allegedly unnecessary surgery was fraud.

The physician and the hospital denied all claims. A neurosurgeon testifying for the physician and hospital said that the surgery was properly performed and according to the standard of care.

The patient claimed medical expenses of $4,856 and wage loss of $28,368. The jury awarded the patient $295,000 compensatory damages and $300,000 punitive damages against the physician only. The physician moved for a new trial.—*Hendrick v. Nork* (Cal.Super.Ct., Sacramento Co., Docket No. 200777, Nov. 10, 1972)

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Council Meeting

Report of Meeting Held September 9, 1973

The Council was called into session at 10:00 AM on Sunday, September 9, 1973, at the Downtown Topeka Holiday Inn. Dr. Thomas F. Taylor, President, called the Council to order.

Present were Dr. Thomas F. Taylor, President; Drs. John N. Blank, Francis T. Collins, Phillip A. Godwin, John W. Travis and Emerson D. Yoder, representing the Executive Committee. Representing the voting Council were Drs. Richard R. Beach, Vernon W. Filley, Richard M. Glover, L. W. Halling, John D. Huff, Ivor H. Jones, W. E. McAllaster, Warren E. Meyer, Alex C. Mitchell, Gerald L. Mowry, Wayne O. Wallace, and Kermit G. Wedel.

Also present were Drs. Murray C. Eddy, Robert E. Feighny, H. Thomas Gray, Robert P. Hudson, Edward L. Jones, Robert H. Kurth, Leo McKee, Herbert C. Modlin, Walter C. Niederee, Lucien R. Pyle, and Charles R. Svoboda. Also present were Dave Morrison, Martha Hunt, Gary Rexford, Jim Agin, Jerry Slaughter, and Oliver E. Ebel.

The minutes of the May 9 meeting were approved.

It was announced that the Society had presented its plea for a single state-wide PSRO before representatives of the regional HEW office. It is anticipated that the area announcements will be made in December.

The Council was reminded that the individual Councilor will act as director for his area for the purposes of Kansas Foundation until an appointment is made. The Foundation now numbers approximately 1,400 members.

The annual Treasurer's report and budget was approved. The Council set the dues for 1974 at $125.00.

Plans for a Leadership Conference were presented. This will be held in February 1974, at the Crown Center, Kansas City, Missouri. The purpose of this meeting will be to involve the unstructured societies, and to help the leadership in those organizations.

The following resolution, endorsing AMA Medicredit Bill, was adopted by the Council, to be presented before the House of Delegates in November:

WHEREAS, There are Americans who either because of their economic status or due to the nature of their illness find the cost of health care to bring financial distress; and

WHEREAS, It appears that the federal government will institute some form of national health insurance; therefore be it

Resolved, That the Kansas Medical Society strongly urge that national health insurance provide a greater benefit for persons of lesser economic stature; and be it further

Resolved, That the AMA sponsored Medicredit Bill does this more equitably than any other legislation currently before Congress; and be it further

Resolved, That the Kansas Medical Society endorse and urge the passage of the Medicredit Bill; and be it further

Resolved, That copies of this resolution be sent to the Kansas Senators and Representatives.

Concerning the question of relations with the State Board of Health, the Council adopted the following resolution for submission to the House of Delegates in November:

Resolved, That the Kansas Medical Society will take no position whatever with reference to the State Board of Health.

On the question of One PSRO for Kansas, the Council adopted the following resolution for submission to the House of Delegates in November:

WHEREAS, PSROs are required by law; and

WHEREAS, The Kansas Foundation for Medical Care was established for the purpose of providing peer review such as required under a PSRO; and

WHEREAS, There are many valid arguments favoring a single PSRO for this state; therefore be it

Resolved, That the Kansas Medical Society strongly urges HEW to appoint the Kansas Foundation for Medical Care as the independent contractor to perform professional standards review as required for implementation of PL 92-603 in Kansas.

A committee, consisting of Drs. Kenneth L. Graham, M. Robert Knapp and Lucien R. Pyle, was appointed for the purpose of formulating a Statement of Society Policies.

A report was made on the visits Mr. Agin and Mr. Slaughter had with the physicians over the state. This program is for the purpose of acquainting the two newest staff members with the physicians, and also to bring the state society closer to them.

It was reported that the Woman's Auxiliary is planning to hold a Health Care Conference, probably February 28-March 2.

Kansas Blue Shield is attempting to sell physicians on the concept of organizing HMPs modeled after an experiment in Wisconsin. The physicians are urged to think about this matter carefully before signing.

It was announced that the Fall meeting of the House of Delegates will be held on Sunday, November 18, 1973, at the Ramada Inn, Manhattan.

Resolutions

To Be Introduced at the Fall House of Delegates, November 18, 1973

RESOLUTION NO. F73-1

(Submitted by the Executive Committee)

Medicredit Bill

WHEREAS, There are Americans who either because of their economic status or due to the nature of their illness find the cost of health care to bring financial distress; and

WHEREAS, It appears that the federal government will institute some form of national health insurance; therefore be it

Resolved, That the Kansas Medical Society strongly urge that national health insurance provide a greater benefit for persons of lesser economic stature; and be it further

Resolved, That the AMA sponsored Medicredit Bill does this more equitably than any other legislation currently before Congress; and be it further

Resolved, That the Kansas Medical Society endorse and urge the passage of the Medicredit Bill; and be it further

Resolved, That copies of this resolution be sent to the Kansas Senators and Representatives.

RESOLUTION NO. F73-2

(Submitted by the Executive Committee)

Kansas State Board of Health

Resolved, That the Kansas Medical Society will take no position whatever with reference to the State Board of Health.

RESOLUTION NO. F73-3

(Submitted by the Executive Committee)

One PSRO For Kansas

WHEREAS, PSROs are required by law; and

WHEREAS, The Kansas Foundation for Medical Care was established for the purpose of providing peer review such as required under a PSRO; and

WHEREAS, There are many valid arguments favoring a single PSRO for this state; therefore be it

Resolved, That the Kansas Medical Society strongly urges HEW to appoint the Kansas Foundation for Medical Care as the independent contractor to perform professional standards review as required for implementation of PL 92-603 in Kansas.

RESOLUTION NO. F73-4

(Submitted by Jack R. Cooper, M.D.)

Kansas Neurosurgical Society

WHEREAS, The neurosurgeons of Kansas wish to be recognized as a component organized specialty society of the Kansas Medical Society; therefore be it

Resolved, That the By-Laws be amended to add Section 4.58, 17, the "Kansas Neurosurgical Society."

RESOLUTION NO. F-73-5

(Submitted by the Southeast Kansas Medical Society, Henry K. Baker, M.D., Delegate)

PSRO and HMO

WHEREAS, The PSRO and HMO laws are in violation of the U. S. Constitution; and

WHEREAS, Legal action is now being brought to test this matter; therefore be it

Resolved, That the Kansas Medical Society, including doctor members and delegates, waste no time at the November meeting planning or discussing these laws.

Journal on Microfilm

Microfilmed copies of current as well as all back issues of the JOURNAL are available through University Microfilm Services, a subsidiary of Xerox Corporation. The 35 mm film fits all standard viewers and provides the JOURNAL in miniature at a savings on binding and storage costs. Write for information or send orders direct to University Microfilm Services, 300 North Zeeb Road, Ann Arbor, Michigan 48106.

KMS Obituaries

HARRY W. KING, M.D.

Dr. Harry W. King, 83, of Kansas City, died August 29, 1973. He was born July 16, 1890, in Iowa.

Dr. King was graduated from the University Medical College of Kansas City in 1913. He practiced medicine in the Kansas City area for 53 years.

Surviving Dr. King is his wife.

WILLIAM W. ORRISON, M.D.

Dr. William W. Orrison, 52, of Meade, died August 29, 1973. He was born August 31, 1921, in Brunswick, Maryland.

Dr. Orrison was graduated from the University of Maryland School of Medicine in 1946.

Survivors include his wife, a son, and two daughters. A memorial fund has been established.

ELWYN S. SHONYO, M.D.

Dr. Elwyn S. Shonyo, 62, of Claflin, died August 13, 1973. He was born May 24, 1911, in Lyndon.

Dr. Shonyo was graduated from the Rush Medical College in 1937.

Survivors include his wife and two sons. A memorial fund has been established.

MARVIN O. STEFFEN, M.D.

Dr. Marvin O. Steffen, 66, of Great Bend, died August 18, 1973. He was born May 24, 1907, in Ellinwood.

Dr. Steffen was graduated from the University of Kansas School of Medicine in 1940. He had practiced in Great Bend since that time.

Surviving Dr. Steffen are his wife, a daughter, and two sons.

Personalities—IN KANSAS MEDICINE

A career Army doctor, who grew up in Norton, has been confirmed by the Senate as Surgeon General of the U. S. Army. He is Richard R. Taylor, brother of **Thomas F. Taylor,** Salina.

Nellie G. Walker, Kansas City, announced her resignation as director of the local health department. She had joined the department in 1955.

Participating in the graduation exercises of St. Vail School of Radiologic Technology were **Millard Spencer** and **Robert C. Lawson,** Topeka.

Governor Docking has announced the following appointments: **E. Raymond Gelvin,** Concordia, Joint Council on Recreation; **Edward F. Steichen,** Lenora, Capitol Area Planning Commission; **C. J. Cavanaugh,** Great Bend, and **George D. Marshall,** Colby, State Board of Healing Arts.

Francis J. Bice, Wakeeney, has announced his retirement from the practice he began here in 1946.

William Nice, Topeka, vice president of the Diabetes Association, was the principal speaker at a recent meeting of the association.

Lucille C. Stephenson, St. Francis, and **Leland C. Reitz,** Manhattan, were the featured speakers for programs on arthritis.

Also retiring from active practice are **Harold L. Hopke,** Shawnee Mission, and **Reynold R. Maixner,** Leoti.

MEDICAL ASSISTANTS CERTIFIED

A record number of 356 medical assistants have become Certified Medical Assistants (CMAs) by successfully passing the 1973 certification examination conducted by the American Association of Medical Assistants (AAMA). A total of 820 candidates sat for the examination given at 66 test centers.

AAMA inaugurated the certification program in 1963, as part of its goal to improve professional standards and education in the field. To date, more than 1200 medical assistants have received certification, their achievement being recognized by use of the letters CMA following their name in professional use.

The certification examination is held annually on the fourth Friday in June. Pertinent information may be obtained from the AAMA office, 1 East Wacker Drive, Chicago 60601.

Following are the names of the 1973 CMAs from Kansas: Betty Alspaw, Wichita; Barbara Bopp, Wichita; Valeda Burris, Wichita; Helen Hartman, Topeka; Marie Hildebrand, Kansas City; Verlene House, Salina; Catherine Jacobs, Gas; Margery Lawrence, Topeka; Peggy Madden, Liberal; Edith Mathes, Topeka; Juanita Mensch, Augusta; Thelma Moody, Hutchinson; Lois Morrissey, Liberal; Blanche Van Hoozer, Yates Center; Pamela Wilson, Wichita.

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

The D. C. Line

Editor's Note: The Editorial Board feels that the Kansas Medical Society would profit by maintaining contact with Representative Bill Roy, both as a source and interpreter of political activities on the national medical front. Accordingly, we are initiating a monthly column written by Dr. Roy. It is hoped that this will not only provide the service intended, but invite a response of readership either through the JOURNAL or to Dr. Roy directly.

It is important that I talk to practicing physicians whenever possible.

This conclusion was greatly reinforced by my conversations with physicians during the August recess. I found again and again that there are substantial numbers of physicians who find no need for health care legislation, and who firmly believe that any federal health care legislation will be detrimental to them and their patients. For example, I asked one group of physicians how many thought Medicare should be discontinued. A substantial majority raised their hands.

First, many contend there is no need for legislation. "You show me anyone in this community or this county who is denied medical care. Doctors have never denied anyone medical care."

Are there people who are too proud to seek medical care because they cannot pay? "Maybe, but I don't know of any; show me one."

What about the uninsurables—those who cannot buy health insurance protection at any price? "They are very few in number and certainly do not provide the excuse for massive federal intervention."

Are not more and more people being priced out of the health care market? There were three frequent answers: (1) No, everything else is going up just as fast; (2) Yes, but this is because of government programs. Stop Medicare and Medicaid and do something about malpractice suits and rapidly increasing costs will stop; (3) Yes, but plumbers cost a lot of money too, and the government does not subsidize the payment of plumbing bills.

There are some physicians who recognize that there are some unmet needs, but they insist that we should let those who know most about medical care—the doctors—solve the problems.

The question—what about the old people and the poor, how should they receive care—received several answers: (1) They should be interested enough in themselves to buy health insurance; (2) The old clinics worked pretty well and they were a lot less expensive; (3) The county used to take care of them okey; (4) Let the poor people go to work; they are poor because they won't work.

In response to the question, is there a need for peer review and utilization review, I heard the following answers: (1) Sometimes, but let local physicians decide if it is needed, and if it is needed, local doctors should do it; (2) No, physicians aren't crooks—leave them alone and they will do the best thing for their patients; (3) No, all we need to do is return to the old ethics and the old ways.

One physician said in response to the question, can we return to the old ethics and the old ways in this day of scientific medicine, "Sure we can. I make house calls—$25, cash on the barrelhead, and it is unreported income."

Two other statements were heard during my travels. A 28-year-old physician said: "If there is national health insurance, I will quit practice."

"Any kind of national health insurance?"

"Yes, any kind of national health insurance."

A doctor's wife said: "My son did not get into medical school because the medical center is against doctors' sons because they (the sons) believe in private practice and fee-for-service medicine."

The setting down as accurately as I can of the most frequent and the most typical answers that I received from Kansas physicians during the recess does not imply that I have simple, ready-made answers to each of the questions. If you infer that I do not agree with most of the answers, you are correct. But I do not say that there is not a kernel of truth in any of the answers.

After one session especially dominated by answers to statements like those above (including the majority opinion for the repeal of Medicare), a reporter who had been there asked me: "What hope is there for satisfactory answers to medical care problems if all doctors think like this?"

The answer is, of course, that all doctors don't think like this.

Benefit Performance

(Continued from page 385)

for the ill, the aged, the convalescent. Someone else must be paid to do it, and the cost is added to the medical bill.

Even though informational projects by the medical profession are often suspect in the eyes of the public as self-serving, there is probably no other group which will confront the public with these points of health care cost. We must, therefore, make them a positive and integral part of our efforts to influence the direction of change in health care delivery. If the public wants a voice in these changes, it must have a realization of the ways in which it has contributed to the present state of the problem.—*D.E.G.*

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VOL. LXXIV
NO. XI

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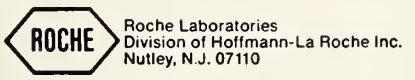

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Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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Volume LXXIV, Number 11—November, 1973

The JOURNAL of the KANSAS MEDICAL SOCIETY

Contents for November

Special Issue: American College of Physicians

Scientific Articles:

Unusual Atrial Arrhythmia: Multifocal Atrial Tachycardia—Merle R. Bolton, Jr., M.D., Wayne Gray, M.D., Raymond Schwegler, M.D. and H. Daniel Lewis, Jr., M.D., Kansas City 394
Glucose Tolerance Tests: Routine Assay of Insulin—Sharfuddin Shah, M.D., Charles R. McReynolds, M.D. and Robert P. Stoffer, M.D., Halstead 399
Renal Transplantation: Complications and Outcome—Robert D. Porter, M.D., Dennis A. Diederich, M.D., Jared J. Grantham, M.D., Creighton A. Hardin, M.D., Arlo S. Hermreck, M.D., Winston K. Mebust, M.D. and Donald R. Tucker, M.D., Kansas City, Kansas 406
Practical Coronary Surgery: Its Place in the Community Hospital—Sherman M. Steinzeig, M.D., Franklin G. Bichlmeier, M.D., William B. Toalson, M.D. and Thomas V. Thomas, M.D., Kansas City, Kansas 411
Persistent Ventricular Tachycardia: The Use of Diphenylhydantoin—Chih-Ping Yang, M.D., Topeka 418
Reflux Esophagitis: A Comparison of Old and New Medical Management—A. Eugene Grossmann, M.D., Arthur P. Klotz, M.D., F.A.C.P., James B. Rhodes, M.D. and Thomas Korb, M.D., Kansas City, Kansas 423

Miscellaneous

Advertisers 11
Announcements 439
Classified Advertisements 11
D. C. Line 433
Editorial Comment 426
HMO Legislation 436
House of Delegates, Resolutions 428
Information for Authors 416
KUMC Postgraduate Education 435
Leadership Conference 422
Midwest Cancer Conference 434
New Members 421
Officers, Councilors 440
Personalities 431
President's Message 425
PSRO Internal Problems 436
Woman's Auxiliary 432

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American College of Physicians Issue

The November issue of the JOURNAL is devoted entirely to papers presented at the Kansas Regional Meeting of the American College of Physicians on February 22, 1973, in Wichita.

The meeting as usual had an attendance of nearly two hundred members and friends. The program chairman was Lew W. Purinton, M.D., and the chairman of arrangements was Richard W. Spann, M.D.

Dr. Purinton has made the collection and preparation of these papers possible. For this, the Editorial Board is deeply grateful.

The six such previous issues of the JOURNAL have been extremely well received and the JOURNAL appreciates this yearly opportunity to expand the audience for these interesting and instructive papers which originated in Kansas.

Scientific ARTICLES

Unusual Atrial Arrhythmia

Multifocal Atrial Tachycardia

MERLE R. BOLTON, JR., M.D., WAYNE GRAY, M.D.,
RAYMOND SCHWEGLER, M.D. *and*
H. DANIEL LEWIS, JR., M.D., *Kansas City*

THE PURPOSE of this paper is to review the clinical, electrocardiographic, and pathologic findings observed in 47 patients with multifocal atrial tachycardia (MAT). Certain predisposing factors in the genesis of this rhythm disturbance will be discussed.[1-10]

Materials and Methods

The electrocardiographic criteria described by Shine, Kastor and Yurchak were used for the diagnosis of MAT.[4] These include an atrial rate greater than 100 per minute, well organized, discrete P waves of varying morphology from at least three different foci, irregular variation in the PP interval, and an isoelectric baseline between P waves. The rhythm strip in *Figure 1* is typical of MAT and demonstrates the electrocardiographic findings described.

Electrocardiograms meeting the criteria mentioned were selected from 26,000 tracings during the course of routine interpretation over the past 36 months. The medical records, serial electrocardiograms, and autopsy data (where available) were reviewed on 47 patients who had at least one episode of multifocal atrial tachycardia.

A relatively unusual atrial arrhythmia, variously referred to as repetitive multifocal paroxysmal atrial tachycardia, chaotic atrial tachycardia, multifocal atrial dysrhythmia, and multifocal atrial tachycardia, has received limited attention in the medical literature over the past several years. Multifocal atrial tachycardia is a unique and important supraventricular arrhythmia.

Results

The 47 patients in this group were all males who were hospitalized at the Kansas City, Missouri Veterans Administration Hospital. The average age of the patients was 70 years, with a range of 46-83 years. This is a relatively unusual arrhythmia, since only 47 of 26,000 electrocardiograms met the diagnostic criteria for MAT. It is an important arrhythmia and associated with a poor prognosis, since 31 of the 47 patients (66%) died during the hospitalization in which this cardiac arrhythmia occurred.

Presented at the annual meeting of the Kansas Chapter, American College of Physicians, Wichita, Kansas, February 23, 1973.

Address reprint requests to: H. Daniel Lewis, Jr., M.D., Department of Medicine, Section of Cardiology, Kansas City V.A. Hospital, 4801 Linwood Blvd., Kansas City, Missouri 64128.

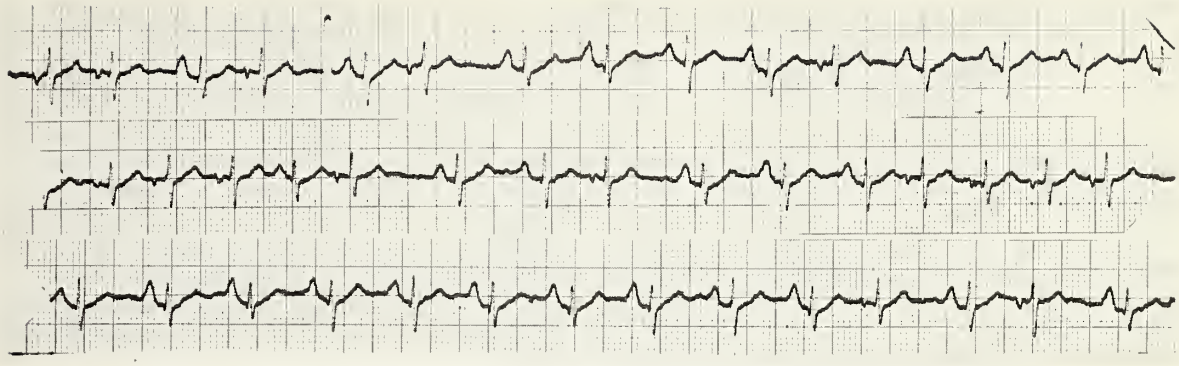

Figure 1. Lead AVF. Rhythm strip demonstrating typical multifocal atrial tachycardia.

Predisposing Factors. Analysis of the clinical records of the 47 patients reconfirmed the impression that this arrhythmia occurs in the setting of an acute or chronic illness of a serious nature *(Table 1)*. The patients invariably had more than one illness, but cardiopulmonary disease, electrolyte and acid base abnormalities, and pharmacologic agents seemed to be particularly important in the genesis of MAT.

Cardiopulmonary Disease. Supraventricular arrhythmias are common in patients with severe respiratory disease.[11, 12] Of our 47 patients with MAT, 29 were significantly disabled by the symptoms of chronic obstructive pulmonary disease. Of the 47 patients, 24 were clinically thought to have arteriosclerotic heart disease. Approximately one half of our patients had typical historical, physical, and radiographic evidence of congestive heart failure. Twelve patients had clinical, radiographic, and bacteriologic evidence of pneumonia. Seven patients had clinical evidence of major pulmonary thrombo-emboli or infarction. An important group of patients were noted to have multifocal atrial tachycardia during an episode of postoperative respiratory failure.

TABLE I

PRINCIPAL COEXISTING CLINICAL DIAGNOSES IN 47 PATIENTS WITH MAT

Disease	*No. of Patients*	*Per Cent*
Chronic obstructive pulmonary disease	29	62
Arteriosclerotic heart disease	24	51
Congestive heart failure	24	51
Pneumonia	12	26
Cerebrovascular insufficiency	9	19
Pulmonary embolus or infarction	7	15
Postoperative respiratory failure	7	15
Acid-peptic disease	6	13
Valvular heart disease	4	9
Carcinoma of lung	3	7
Acute myocardial infarction	2	4

From this data it seems apparent that underlying cardiopulmonary disease is an important common denominator in the clinical setting of those patients who experience MAT. Of additional interest is the fact that significant valvular heart disease was noted in only four of our patients, and that MAT occurred in the course of acute myocardial infarction in only two patients. Burch,[6] in his series of 31 patients with chaotic atrial mechanism noted an unusually high incidence of coexisting diabetes mellitus. An unusual incidence of glucose intolerance was not observed in our group of patients, and has not been a consistent finding by others.[8]

Electrolyte and Acid Base Abnormalities. The electrolyte and acid base status at the time MAT developed was subsequently analyzed in our patients. Of the 47 patients, 42 had a serum potassium determination performed on the day the rhythm disturbance was first detected *(Table 2)*. Whereas most of the patients were found to have a serum potassium within normal limits, it is noteworthy that 13 patients were significantly hypokalemic.

Arterial blood gas determinations were similarly available in 31 of 47 patients *(Table 3)*. Only five patients were found to have an arterial blood pH within the range of normal, and only three patients were found to be acidotic. Twenty-three patients (74%) were found to be significantly alkalotic. Analysis of the acid base status of patients with cardiopulmonary disease and ventilation perfusion imbalance, who were often being treated with corticosteroids, diuretics and restricted salt diets, is difficult. However, respiratory alkalosis was the most common acid base disturbance noted.

As might be expected, one half of our patients were receiving oxygen therapy, some in the form of cannula or Campbell mask, and others via nasal endotracheal tube and volume ventilator support. "Inappropriate ventilation" leading to respiratory alkalosis has been suggested as a cause of cardiac

TABLE II

SERUM POTASSIUM IN PATIENTS WITH MAT

Serum Potassium Value	*No. of Patients*	*Per Cent*
Potassium < 3.5 mEq/L	13	31
Potassium 3.6-4.5 mEq/L	22	52
Potassium 4.6-5.3 mEq/L	7	17

arrhythmias by Ayres and Grace.[13] That such a mechanism may be important in the genesis of MAT will be commented upon subsequently.

Sixteen of 31 patients had a Co_2 greater than 60 mm Hg, and 15 patients had significant hypoxia with a Co_2 of 60 mm Hg or less. Only six patients were noted to have significant carbon dioxide retention.

Pharmacologic Agents. Certain drugs have been incriminated in the genesis and perpetuation of MAT. For this reason, particular attention was paid to the medications and therapeutic agents being administered to our patients at the time MAT developed *(Table 4)*.

Not unexpectedly, we found that 41 of 47 patients were receiving a digitalis preparation at the time MAT was noted. Chung[8-9] has suggested that digitalis intoxication is a common cause of multifocal atrial tachycardia. This remains a possibility, because the incidence of supraventricular arrhythmias in patients with respiratory failure has been estimated to be approximately 5 per cent.[12] Atrial tachycardia, and atrial tachycardia with block are often associated with digitalis therapy.[12, 11] It has been suggested that the hypoxia accompanying respiratory failure sensitizes the myocardium to digitalis.[12] Despite these factors, it has been difficult for us to clearly substantiate the role of digitalis intoxication in the genesis of multifocal atrial tachycardia. Other investigators have also doubted the role of digitalis in precipitating this rhythm disturbance.[4, 5, 10] Only one elderly man in our group of patients clearly developed MAT secondary to digitalis intoxication, and he was hypokalemic while receiving digitoxin 0.2 mg per day. Against the suggestion that digitalis intoxication is an important cause of this arrhythmia is the fact that MAT often subsides while maintenance doses of digitalis are continued. The rarity in our group of patients of other arrhythmias which are commonly associated with digitalis intoxication also raises doubt as to the role of digitalis in precipitating MAT. For example, only four patients in our group experienced paroxysmal atrial tachycardia with block, multifocal premature ventricular contractions, or bigeminal rhythm.

TABLE III

ACID BASE STATUS OF 31 PATIENTS WITH MAT

Arterial Blood Gas Factor	*No. of Patients*	*Per Cent*
pH 7.35-7.45	5	16
pH < 7.35	3	10
pH > 7.45	23	74
Po_2 > 60 mm	16	51
Po_2 60 mm or below	15	49
Pco_2 > 49 mm	6	19
Pco_2 49 mm or below	25	81

TABLE IV

MEDICATIONS AND THERAPEUTIC AGENTS ADMINISTERED TO 47 PATIENTS AT THE TIME MAT DEVELOPED

Medication	*No. of Patients*	*Per Cent*
Digitalis preparations	41	87
Bronchodilators (Aminophylline, Isuprel, Ephedrine)	26	55
Diuretics	12	26
Sedatives	12	26
Oxygen	24	51

Bronchodilators were commonly administered to our patients because of the high incidence of underlying chronic lung disease. The administration of these sympathomimetic agents may have aggravated the MAT by contributing to the rapid atrial rates, as previously mentioned by Yurchak.[4]

Diuretic preparations were administered to approximately one fourth of our patients and probably contributed to the hypokalemia noted in some. Sedative preparations were being administered to 12 of our patients at the time MAT developed. That sedation may be a significant factor in the deterioration of ventilatory performance in patients with severe chronic obstructive pulmonary disease (COPD) is well known.

Electrocardiographic Review. Serial electrocardiographic review revealed that many of our patients experienced a number of associated arrhythmias either preceding or following an episode of MAT *(Table 5)*. For example, 25 of 47 patients had another atrial arrhythmia during their hospital course in addition to MAT (excluding sinus tachycardia and atrial premature beats). Disturbances of atrioventricular conduction were also noted in the form of first degree AV block. Junctional tachycardia was seen in three patients. Ten patients were noted to have intraventricular conduction defects at some time during their hospitalization, 20 patients were observed to have

TABLE V

ASSOCIATED ARRHYTHMIAS EITHER PRECEDING OR FOLLOWING MAT

Arrhythmia	*No. of Patients*
Atrial Arrhythmias	
Atrial premature beats	19
Wandering atrial pacemaker	9
Paroxysmal atrial tachycardia	2
Atrial flutter	4
Atrial fibrillation	11
Disturbances of Atrioventricular Conduction	
First degree AV block	8
Junctional rhythm	3

ventricular premature beats, and three patients experienced ventricular tachycardia. Rather characteristically, an episode of MAT was noted to persist for a period of a few hours to several days. No patient was followed who had MAT on a chronic basis.

Associated electrocardiographic findings were also evaluated by serial review. Of particular interest is the frequency of those findings associated with chronic lung disease, position changes consistent with diffuse pulmonary disease: right atrial overload, low voltage, right axis deviation, right ventricular hypertrophy, and right bundle branch block *(Table 6)*. Left atrial overload and left ventricular hypertrophy were also noted rather commonly.

Autopsy Data. Thirty-one of 47 patients died during the hospitalization when MAT was first detected. Post-mortem examinations were performed in 17 patients, and helped substantiate clinical impressions regarding underlying disease states *(Table 7)*. As noted, severe pulmonary emphysema was found in 76 per cent of our autopsied group of patients. Myocardial hypertrophy and dilatation were seen in

TABLE VI

ASSOCIATED EKG FINDINGS IN 47 PATIENTS

Abnormality	*No. of Patients*	*Per Cent*
ST-T segment changes of ischemia/digitalis	25	53
Position changes consistent with diffuse pulmonary disease	14	30
Right atrial overload	9	19
Low voltage	7	15
Right axis deviation	5	11
Right ventricular hypertrophy	4	9
Right bundle branch block	4	9

TABLE VII

POST-MORTEM FINDINGS IN 17 PATIENTS WITH MAT

Autopsy Finding	*No. of Patients*	*Per Cent**
Pulmonary emphysema	13	76
Myocardial hypertrophy	12	70
Congestive heart failure	9	53
Pneumonia	8	47
Generalized atherosclerosis	8	47
Coronary atherosclerosis	6	35
Pulmonary emboli and/or infarction	6	35

* Of autopsied patients.

12 of 17 patients, and severe pulmonary congestion was noted in nine of 17 patients. Pneumonia was also a common post-mortem finding. Atherosclerosis of significant degree was not as common a finding as expected in this elderly group of patients. The finding of pulmonary emboli involving major pulmonary arterial branches in 35 per cent of the autopsied group of patients is of interest. It is our opinion that the sudden clinical appearance of multifocal atrial tachycardia may prove, on rare occasions, to be an electrocardiographic change accompanying acute pulmonary embolism, just as other supraventricular arrhythmias are often the harbinger of pulmonary embolism.[15]

Discussion

Multifocal atrial tachycardia is a relatively unusual, but important supraventricular arrhythmia. The high mortality rate associated with the presence of this rhythm disturbance reflects the fact that MAT is most frequently encountered in elderly, severely ill patients who are suffering from chronic obstructive lung disease. Congestive heart failure, pneumonia, and major thrombo-embolic episodes are a relatively common part of the clinical presentation in this group of patients. MAT was not uncommon in the post-operative period of patients who had acute respiratory failure for whatever reason.

The mechanism of multifocal atrial tachycardia may be similar to that of unifocal atrial tachycardia,[9] except with several ectopic foci. Alkalosis, hypoxia and hypokalemia may, on occasion, be significant predisposing factors in the genesis and maintenance of this rhythm disturbance. Cardiac arrhythmias have previously been reported by Ayres and Grace,[13] to occur as a result of inappropriate ventilation and hypoxia. The frequency of alkalosis and cardiac arrhythmias is discussed in detail in their review.

It is attractive for us to speculate that hypoxemia, hypokalemia, alkalosis and other, perhaps more subtle

metabolic changes occurring in varying combinations, may underlie the appearance and persistence of multifocal atrial tachycardia. As mentioned, digitalis intoxication per se was difficult for us to incriminate as an etiologic factor.

During our initial experience with this arrhythmia, multiple anti-arrhythmic agents, including lidocaine, diphenylhydantoin, propranolol, quinidine, and procaine amide were tried unsuccessfully. Direct current cardioversion was also tried on several occasions, but without success. The administration of digitalis to those patients in congestive heart failure was an important therapeutic intervention, but the administration of increasing doses of digitalis to control ventricular rate is ill-advised because of the possibility of producing MAT with progressive AV block, or other more serious arrhythmias. Of those patients surviving, meticulous attention was given to the underlying illnesses. As described, many different abnormal parameters were noted to coexist with this arrhythmia, such as low serum potassium, high arterial pH, and the use of bronchodilators. Clinically, attention was focused on the correction of as many of these parameters as possible, and the arrhythmia was noted to improve or disappear, coincidental with improvement in the patient's general status.

It would appear that alteration of the acid base status, particularly respiratory alkalosis, may be the most significant predisposing factor in the genesis of MAT. Specific attention should, therefore, be directed toward adequate oxygenation with proper adjustment of the ventilatory rate to minimize respiratory alkalosis. Electrolyte deficits, such as hypokalemia, should be corrected with appropriate supplements. The use of bronchodilators in patients with MAT should be weighed against the possible aggravation of the underlying rhythm disturbance. Treatment of the underlying disease, specifically chronic obstructive pulmonary disease, is much more beneficial than the use of specific antiarrhythmic agents. Finally, it should be reiterated that the development of MAT is associated with a grave prognosis.

Summary

Clinical analysis of 47 patients with multifocal atrial tachycardia (MAT) was done. Chronic obstructive pulmonary disease was present in 29 patients and constituted the most common underlying disease. Although 41 patients were receiving digitalis at the onset of the arrhythmia, this drug alone did not appear to be a major consideration in the genesis of MAT. Alkalemia occurred in 23 patients coincidental with the development of MAT and may be one of the most important factors in the development of MAT. The occurrence of MAT was associated with a high mortality (31 patients, 66%, died during the hospitalization when MAT was first detected). The best method of therapy for this rhythm disturbance was careful management of the underlying illness. Correction of hypoxemia, alkalemia, and hypokalemia would seem to be of utmost importance in therapy.

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Attend the
HOUSE OF DELEGATES MEETING
See P. 417 for details

Glucose Tolerance Tests

Routine Assay of Insulin

SHARFUDDIN SHAH, M.D., CHARLES R. McREYNOLDS, M.D. *and* **ROBERT P. STOFFER, M.D.,** *Halstead*

The purpose of this study is to determine if the routine measurement of insulin concomitant with the measurement of glucose in all samples of sera obtained in the standard glucose tolerance test would be of value in the diagnosis of diabetes and proper management of treatment.

THE METHODOLOGY used in this study consisted of the measurement of insulin by the radioimmune competitive binding assay, measurement of glucose by the autoanalyzer potassium ferrocyanide reduction method, and plotting these values on a single chart; plotting glucose levels linearly and insulin levels as percent increase in basal level.

One thousand patients were studied in this survey. Patients were included in the survey on the basis of raised fasting blood sugar (FBS), raised two-hour Glucola test, family history of diabetes, or if they had signs and symptoms of diabetes. FBS between 70-110 mg/100 ml was considered to be within normal limits; 110-130 mg/100 ml was considered to be doubtful. FBS of over 130 was considered to be definitely abnormal. Blood sugar levels at one-hour intervals of 160 mg/100 ml, at one and one-half hour 150 mg/100 ml, and at two hours with maximum of 140 mg/100 ml were considered to be within normal range. In consideration of normal values in the glucose tolerance test (GTT), specimens were obtained at one, one and one-half, two, and three hours. An abnormal test was present if the one-, one and one-half-, and two-hour specimens were 185, 160, and 140 mg/100 ml respectively—with all three values above those listed. Also, if FBS one-, two-, and three-hour levels totaled 600 mg/100 ml or above, the individual was considered to have diabetes mellitus.

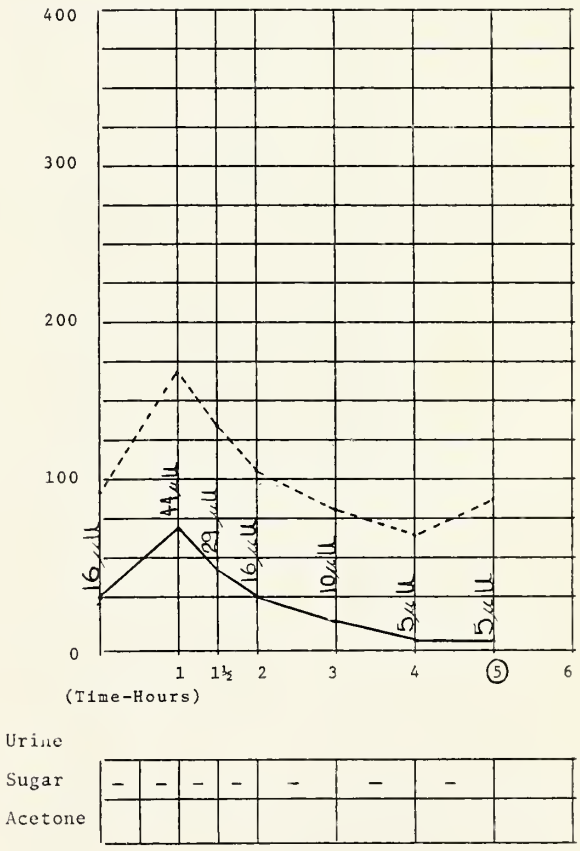
If one of the blood sugar levels was raised, the patient was considered to be a borderline diabetic. Chemical diabetes mellitus was the diagnosis if no symptoms were present.

Case Reports

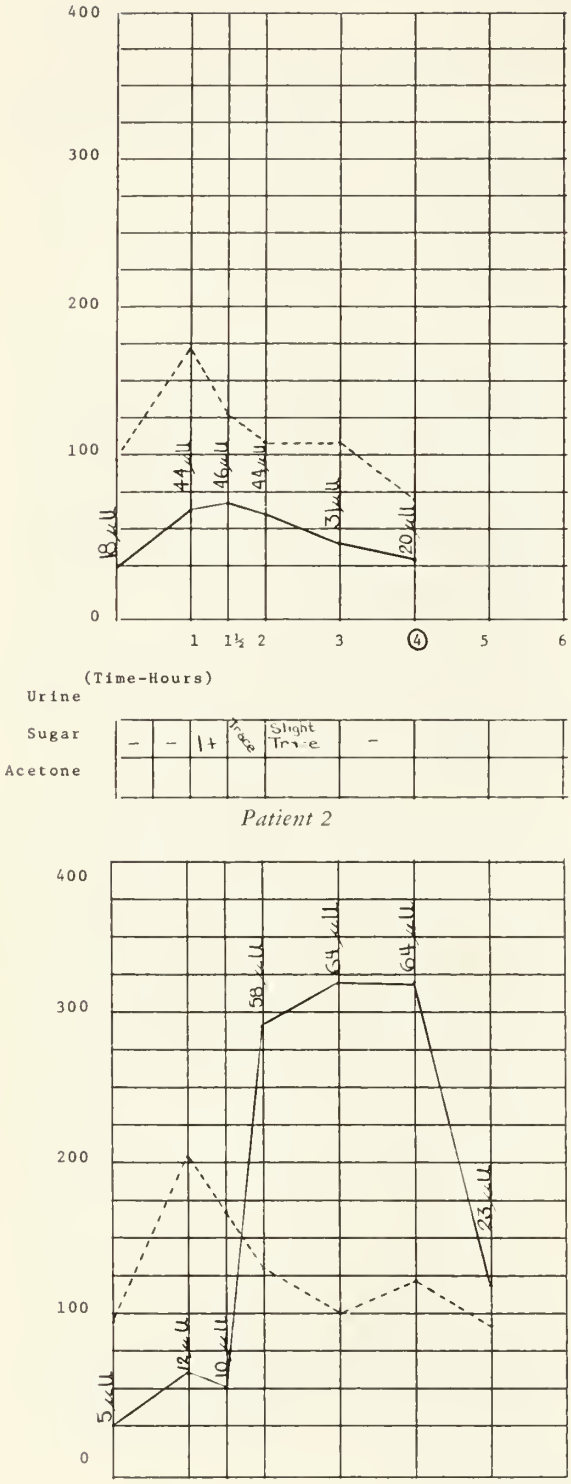
A survey of the results in a number of different patients will now be presented. They are representative of a number of patients in each group and it is our opinion that the insulin assay is somewhat helpful in each case. None of the patients has been a known diabetic, and none had taken insulin or oral antibiotic medication.

Patient 1: A 68-year-old male; weight, 58.1 kg (128 lb.); height, 168.2 cm (5 ft. 6 in.).

This is a normal GTT and shows normal glucose levels on the dotted line and insulin assay in micro units (expressed on the graph as percent of basal insulin level).



Patient 1

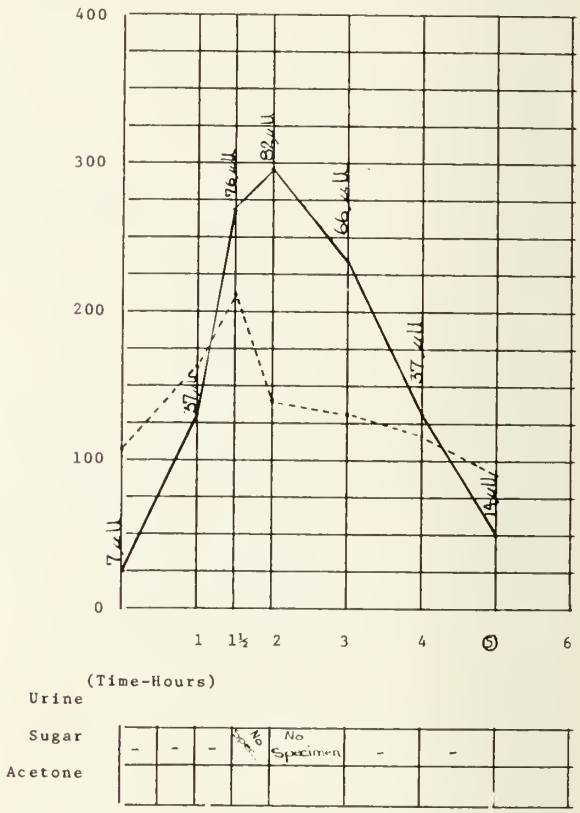


Patient 2

Patient 2: A 67-year-old male; weight, 87.7 kg (193 lb.); height, 168.6 cm (5 ft. 6½ in.).

Normal GTT and insulin curve with a trace of sugar at two and three hours. The one-hour specimen is mildly elevated and may represent a short "spill," or if more pronounced might suggest renal glycosuria.

Patient 3: A 77-year-old female; weight, 62.2 kg (137 lb.); height, 158.4 cm (5 ft. 2 in.).



Patient 3

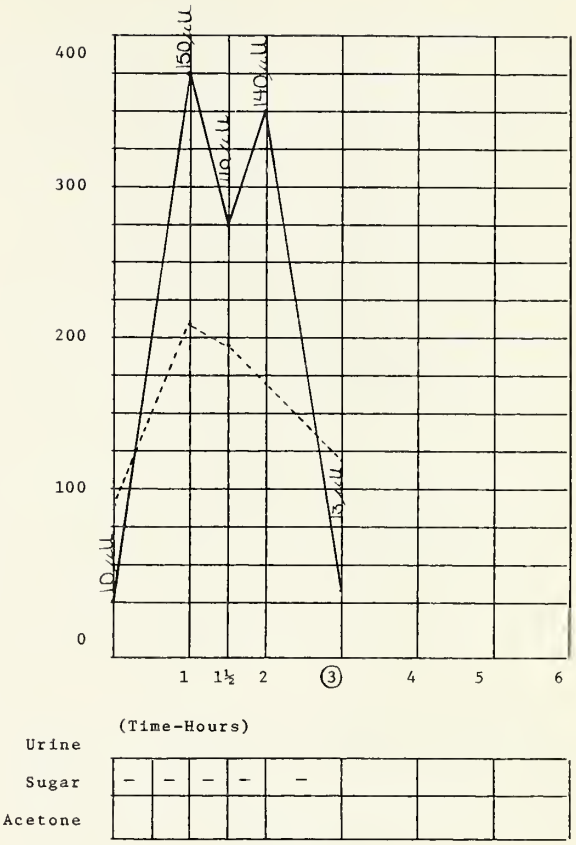
Elevated GTT, especially at one and one-half hours, with other readings borderline. Insulin output is high. The patient is 77 years of age, and has decreased tolerance. No therapy is required, the condition may be regarded as almost normal, considering age.

Patient 4: A 76-year-old male; weight, 59.5 kg (131 lb.); height, 142.1 cm (4 ft. 7 in.).

Elevated GTT, especially at one hour, with high output slightly delayed.

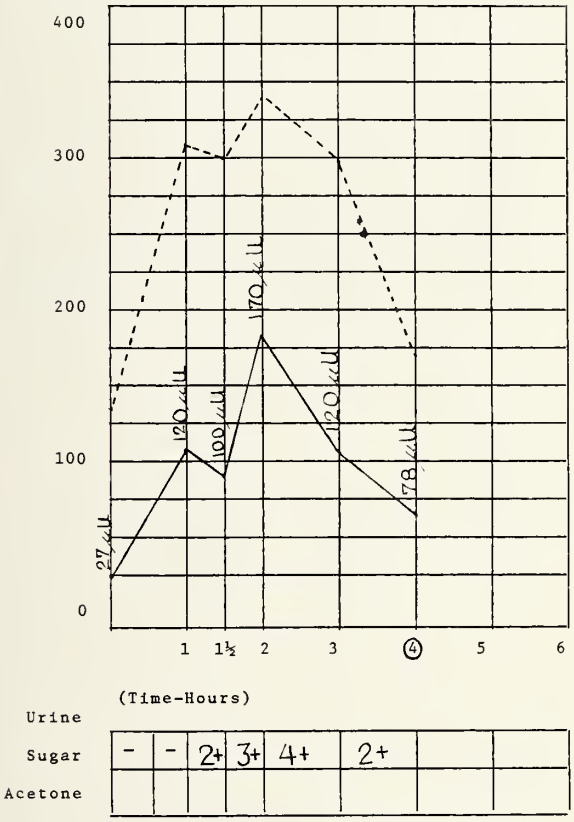
Patient 5: A 74-year-old male; weight, 65.3 kg (143.5 lb.); height, 163.5 cm (5 ft. 4 in.).

Elevated GTT with high insulin output—characteristic of diabetes mellitus, chemical type in this case.



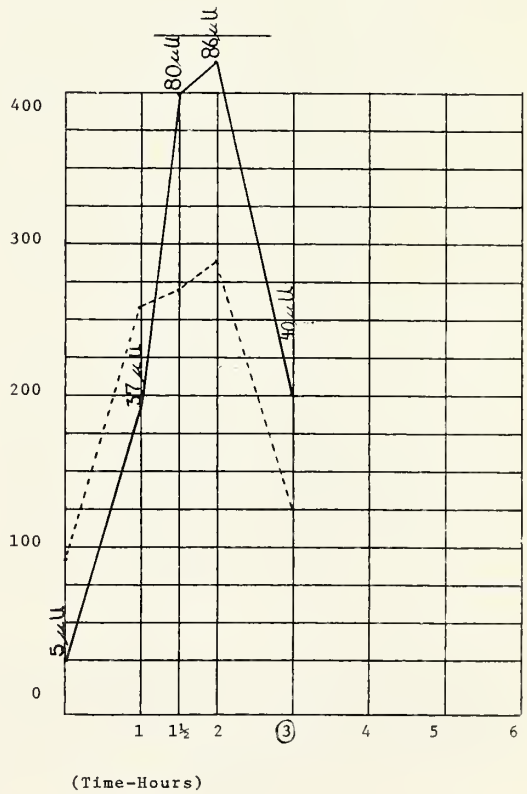
Patient 5

Patient 6: A 70-year-old male; weight, 78.6 kg (173.5 lb.); height, 168 cm (5 ft. 5 in.).



Patient 6

Abnormal GTT, obesity, and high insulin output.

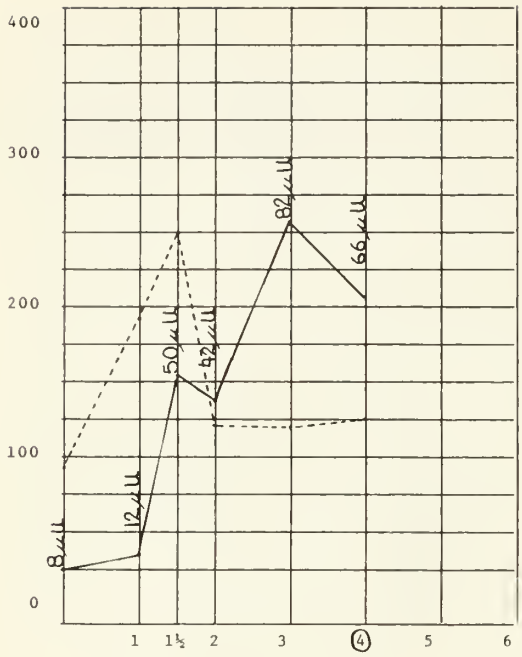


Patient 7: A 67-year-old male; weight, 85.3 kg (188 lb.); height, 158.3 cm (5 ft. 2 in.).

Abnormal GTT with (high values) obesity, high insulin output, and high fasting insulin level.

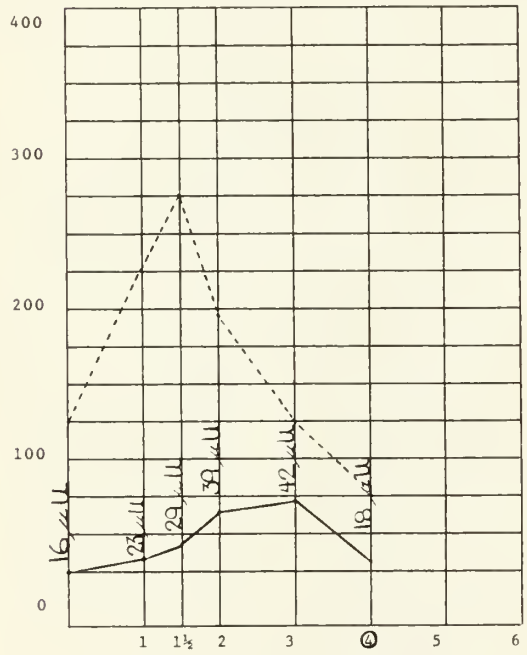
Urine								
Sugar	–	Trace	4+	4+	2+			
Acetone	–	–	–	–	–			

Patient 7



Urine								
Sugar	–	–	3+	1+	–	–		
Acetone	–							

Patient 8



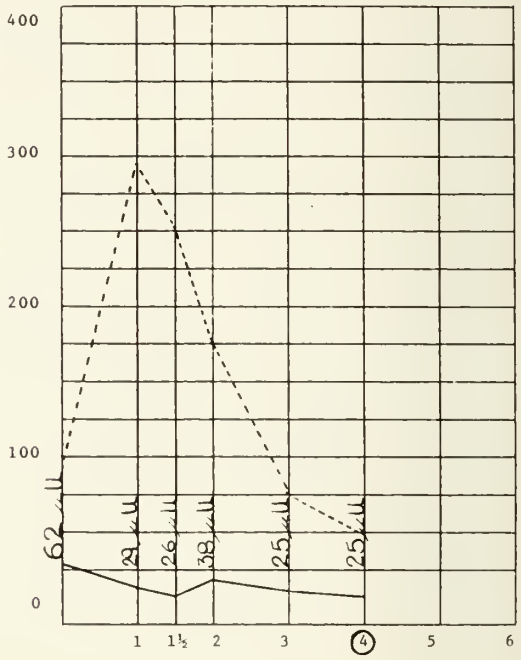
Urine								
Sugar	–	–	–	–	–	–		
Acetone	–	–	–	–	–	–		

Patient 10

Patient 8: A 66-year-old female; weight, 63.1 kg (139 lb.); height, 159.6 cm (5 ft. 2.5 in.).

Abnormal GTT (diabetic) with initially low and later high insulin output (delayed response).

Patient 9: A 63-year-old female; weight, 38.8 kg (85 lb. 4 oz.); height, 158.3 cm (5 ft. 2 in.).



Urine								
Sugar	–	1+	3+	4+	1+	–		
Acetone								

Patient 9

Abnormal GTT with fairly high initial insulin output but no response to glucose elevation and a gradual fall. In considering therapy, insulin would be the treatment agent along with diet. Oral agents would be ineffective.

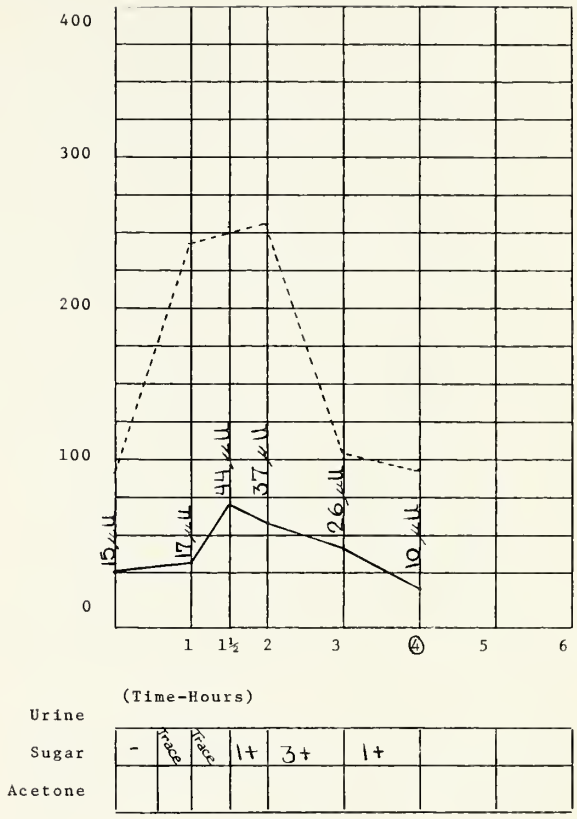
Patient 10: A 47-year-old male; weight, 105.3 kg (232 lb.); height, 185.5 cm (6 ft. 1 in.).

Abnormal GTT with low insulin output in an obese patient with chemical diabetes. Diet would be the treatment of choice.

Patient 11: A 71-year-old male; weight, 93.1 kg (205 lb.); height, 157.1 cm (5 ft. 2 in.).

Another abnormal GTT with low insulin output, considerable glycosuria, and obesity. Diet would be the treatment of choice. Certain patients might be considered for oral agents if not well controlled, although there might be some controversy.

There has been a lot of discussion against the use of oral hypoglycemic agents following the report of a university group diabetic survey, because it has been noted that diabetic patients on oral hypoglycemic agents have had many more cardiovascular and cerebrovascular accidents when compared to those who have been on diet alone or diet plus insulin. The sulfonylurea group is considered better than the biguanide series of oral hypoglycemic agents by the university group diabetic survey, but once these patients become ketotic, they should not have oral hypoglycemic agents; they should be treated with diet and insulin.

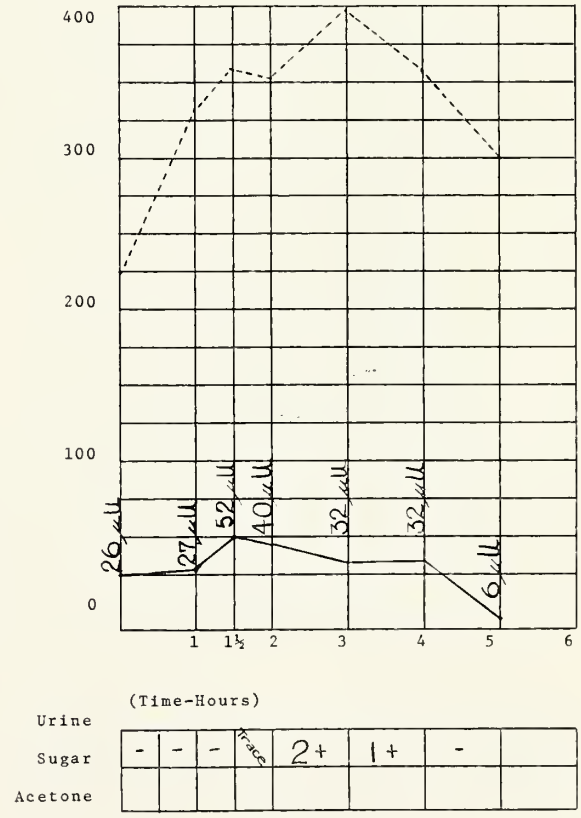


Patient 11

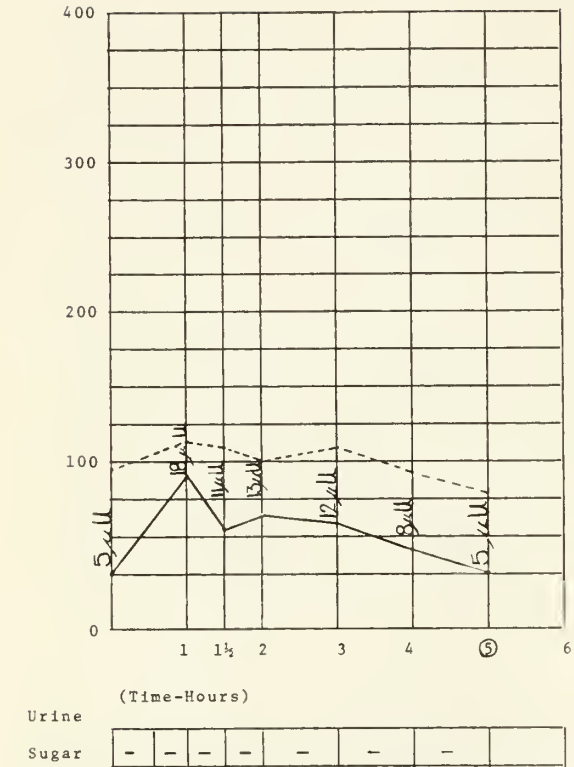
Patient 12: A 56-year-old female; weight, 90.5 kg (198 lb. 8 oz.); height, 163.4 cm (5 ft. 4 in.).

This patient is obese with a high glucose tolerance and very low insulin output. She may respond to diet alone or need insulin. Oral agents would not be expected to be helpful.

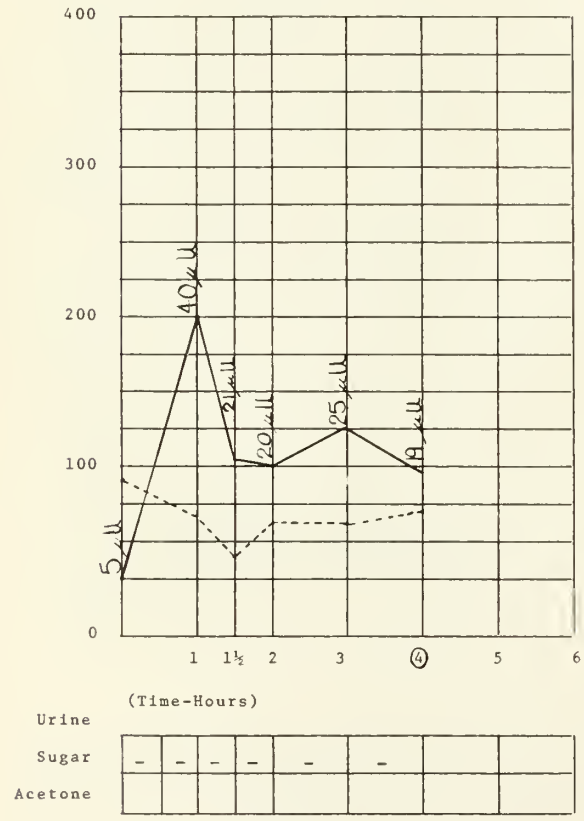
During this study, it was noted that a number of the tests had "flat" GTT curves with varying insulin responses to the oral glucose load.



Patient 12



Patient 13

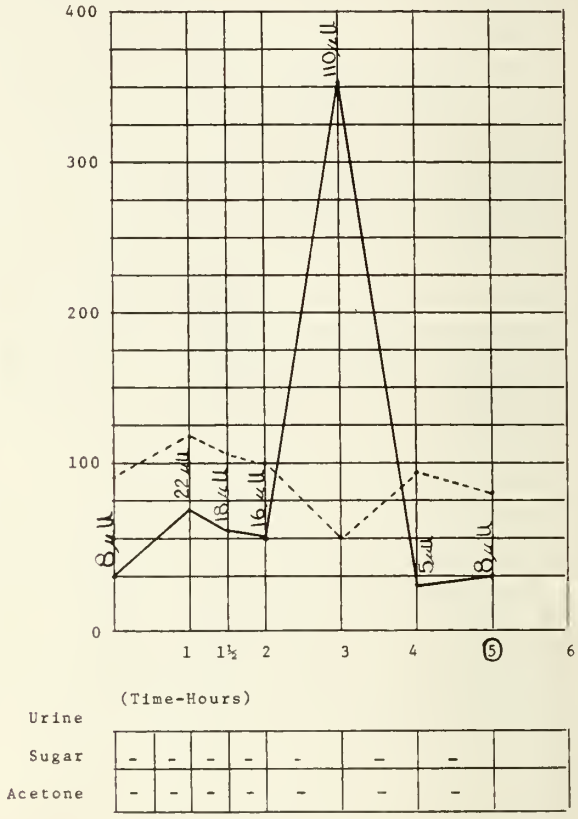


Patient 15

Patient 13: A 32-year-old male; weight, 52.6 kg (116 lb.); height, 171.7 cm (5 ft. 6 in.).

Flat GTT with low output of insulin.

Patient 14: A 27-year-old male; weight, 56.8 kg (125 lb); height, 168 cm (5 ft. 5 in.).



Patient 14

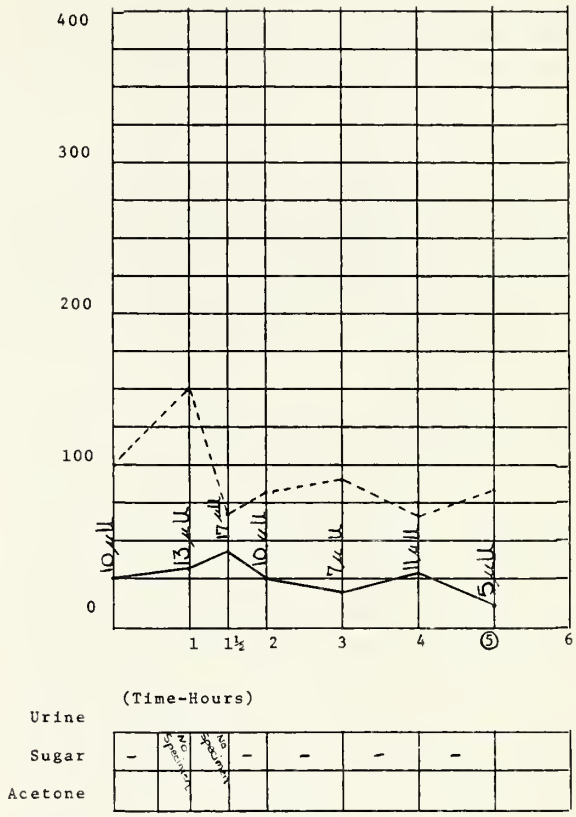
Flat curve with an initial low insulin output but a spiking high value at three hours that occurs at the same time as the dip in blood sugar to 50 mg/100 ml. This can be called reactive hypoglycemia. One might wonder whether the sugar value is correct without the high insulin value.

Patient 15: A 31-year-old female; weight, 47.3 kg (104 lb. 4 oz.); height, 159.6 cm (5 ft. 3 in.).

Flat curve with a low value at one and one-half hours, and an elevated insulin response prior to the low value. One might consider an insulinoma with the use of other appropriate tests (*i.e.,* serial glucose or a tolbutamide test). The immuno-reactive insulin level may be useful in a suspected patient with insulinoma in that the insulin level may rise to exceptionally high levels (up to the 1,000 uU range).

Patient 16: A 50-year-old male; weight, 93.2 kg (205 lb. 12 oz.); height, 177.6 cm (5 ft. 10 in.).

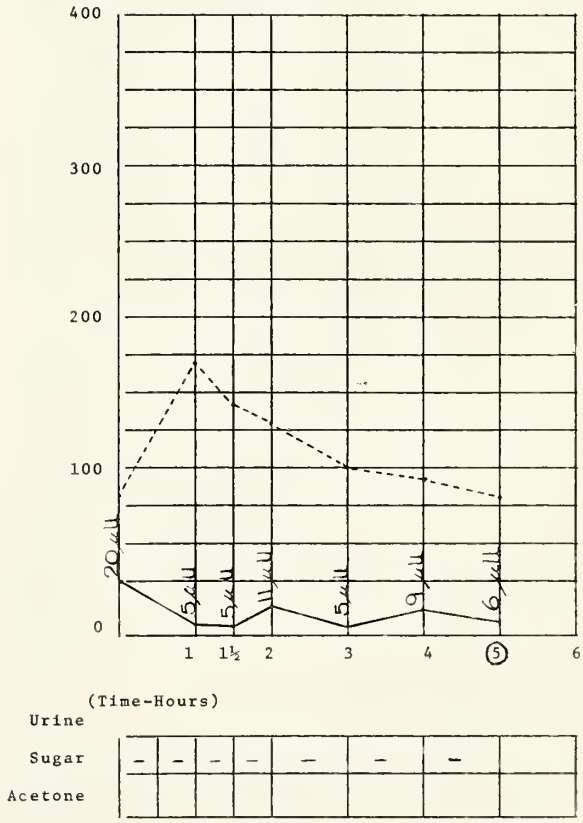
Flat GTT and low insulin output in an obese male.



Patient 16

Patient 17: A 65-year-old female; weight, 67.6 kg (149 lb.); height, 158.3 cm (5 ft. 2 in.).

Normal GTT with low insulin output. It is possible that such patients may develop abnormal glucose tolerance when under stress with disease, infection, and the like.



Patient 17

Summary

As one can see from the above patterns, the immuno-reactive insulin, when tested along with the usual glucose tolerance, does have value as well as interest. It helps to differentiate diabetes with high and low insulin output, as well as minimal output. Obesity is not necessarily associated with high output. Appropriate treatment can be instituted. There are striking patterns associated with reactive hypoglycemia, as well as those with early diabetes and islet cell tumor.

The physician will be guided by the response to treatment in each case, and insulin curves may be of assistance in initial management. We hope that with further study, we can learn more about patients with abnormal glucose tolerance tests.

Renal Transplantation

Complications and Outcome

ROBERT D. PORTER, M.D., DENNIS A. DIEDERICH, M.D., JARED J. GRANTHAM, M.D., CREIGHTON A. HARDIN, M.D., ARLO S. HERMRECK, M.D., WINSTON K. MEBUST, M.D. *and* DONALD R. TUCKER, M.D., *Kansas City, Kansas*

Twenty-eight kidney transplants have been performed at the Kansas University Medical Center. The overall patient survival rate is 91 per cent. Seventy-five per cent of the kidneys transplanted from related donors and 60 per cent of those from non-related cadaver donors have functioned for at least nine months. Serious complications including infections, rejection of the allograft, and acute tubular necrosis generally occurred during the first three months post transplant. Sixteen patients are rehabilitated with unrestricted activity, and eight heads of households are working full time. Renal transplantation is a practical therapy for end-stage renal failure.

KIDNEY transplantation began in the back wards of the Massachusetts General Hospital in 1947. Two surgical residents, Dr. Hufnagel and Dr. Hume, grafted a kidney into the antecubital fossa of a woman dying from acute tubular necrosis. The allograft functioned well enough to sustain her life until her own kidney could take over. In 1954, again in Boston, a kidney was transplanted from a living man to his identical twin brother as experimental therapy for end-stage renal failure, and the dying man survived for eight years.[1] Since then, nearly 10,000 kidney transplants have been done and currently renal transplantation is offered in most medical centers throughout the world.

We began performing kidney transplants at the Kansas University Medical Center in July 1969. Since then, 23 patients have received transplants, and 18 of them have life-sustaining renal function. The problems of kidney transplantation, to which this discussion is addressed, actually begin at the time the prospective recipient is initially evaluated. Not all patients who develop end-stage renal failure will benefit from renal transplantation. The recipient must have a normal lower urinary tract and no evidence of systemic disease such as cancer, diabetes mellitus, or severe arteriosclerosis. Prior to transplantation, the acceptable recipients are supported with chronic hemodialysis at the University Medical Center or at the St. Francis Hospital Dialysis Center in Wichita. In the past year, the Kansas City Veterans Administration Hospital has begun to do transplants as part of our program. Their patients are not included in the present analysis because the followup is too brief.

In the preparation for the allograft, each of the prospective recipients was typed for HLA histocompatibility antigens.[2] Immediately prior to the transplant operation, the recipient's blood was examined to rule out the presence of circulating cytotoxic antibodies.[3]

At the transplant operation, the vascular surgeon anastomosed the renal artery to the iliac artery of the recipient, and the urologist implanted the donor ureter into the recipient's bladder. The post-surgical care and the immunosuppressive therapy were managed by members of the Nephrology Section. Clearly, renal transplantation is a team effort.

Immunosuppression is essential to the success of renal transplantation. As shown in *Table I,* our protocol for immunosuppression includes the use of a constant dose of Azathioprine of 3 mg/kg/day and intravenous methyl prednisolone, 4 mg/kg/day for two days; on the third and fifth days the dose of methyl prednisolone was reduced by 1 mg/kg. The patient was converted to oral prednisone therapy usually after the first week, and the dose of this medication was tapered gradually to a maintenance level after four to six weeks.

For an acute rejection crisis, huge doses of methyl prednisolone, 10 to 15 mg/kg, were administered intravenously every 24 to 72 hours.[4] We also used local irradiation of the graft in several patients.

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TABLE I

IMMUNOSUPPRESSIVE PROTOCOL

First week:	
Azathioprine	3 mg/kg daily
Methyl prednisolone ..	4 mg/kg I.V. day 1, 2
	3 mg/kg I.V. day 3, 4
	2 mg/kg I.V. day 5, 6
	1 mg/kg I.V. day 7
Maintenance:	
Azathioprine	1-3 mg/kg daily
Prednisone, initially reduced by 5 mg/day monthly	1 mg/kg daily
Acute rejection:	
Methyl prednisolone ..	10-15 mg/kg I.V. q. 24-72 hrs.
Local graft irradiation	150 r. q.o.d. X 4

Our overall experience in renal transplantation from July 1969 to February 1973 is shown in *Table II*. We have performed eight transplants from living related donors; six of these transplants carry on life-sustaining renal function. We have performed 20 transplants from cadaver donors into 16 individual recipients. Four of these recipients required retransplantation for reasons to be discussed later. Twelve recipients of cadaver kidneys presently enjoy life-sustaining renal function. One patient, who originally received a living related kidney which was lost to rejection, subsequently received a cadaver kidney, which also failed. In our experience, the overall mortality rate was 8.7 per cent, which compares quite favorably with the national experience of about 33 per cent over the same period of time.[5]

Thus, while kidneys can be transplanted into humans at only moderate risk to the life of the recipient, an important concern is how well the transplanted kidneys fare. The kidney survival curves for living related and cadaver donor transplants are shown in *Figure 1*. The number of months after the transplant

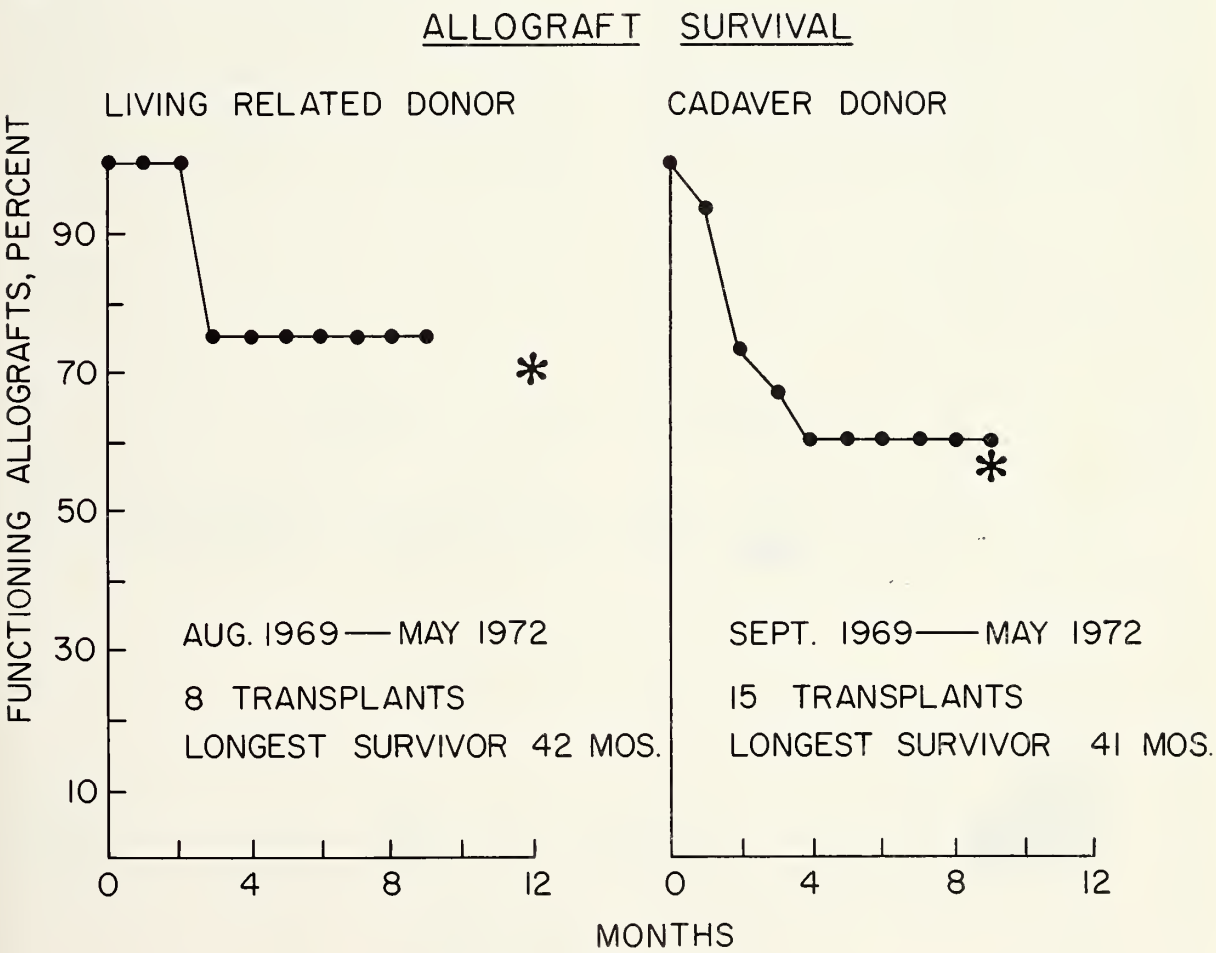

Figure 1. Allograft Survival.

TABLE II

RENAL TRANSPLANT EXPERIENCE
JULY 1969-FEBRUARY 1973

Living related donor	8
Transplant function	6
Patient survival	8
Cadaver donor	20
Individual recipients	16
Patient survival	14
Transplant functioning	12
Mortality	2/23
Mortality	8.7%
National[5]	33 %

TABLE III

MANIFESTATIONS OF REJECTION

Fever
Tender kidney
Swelling of the kidney
Hypertension
Oliguria
Elevation of serum creatinine
Malaise
Low urinary Na concentration

is on the abscissa and the per cent of kidneys functioning adequately is on the ordinate. Only patients who have been followed for at least nine months after the transplant procedure are included in this analysis. The longest period of observation of a recipient of a living related allograft is 42 months; the longest period of observation of a recipient of a cadaver allograft is 41 months. A striking feature is the loss of transplanted organs during the early months in both groups of patients. We lost two kidneys during the first two months in the related living group, after the initial months survival stabilized at about 75 per cent. Several kidneys were lost during the first four months in the cadaver group as well; however, kidney survival stabilized at about 60 per cent. Our survival rates compare favorably with the national experience. The asterisk at nine months on the living related curve indicates the recent, national survival figure of 70 per cent. For cadaveric allografts, the figure is 58 per cent at one year.[5] It appears that the high risk period for the grafted kidneys is during the first four months after the transplantation.

It is well known that rejection is one of the major complications jeopardizing the function of the allografted kidney. In the present study, allograft rejection was diagnosed when we detected a significant number of the signs and symptoms shown in *Table III*. Fever, tenderness of the kidney, enlargement of the kidney, hypertension, oliguria, rising serum creati-

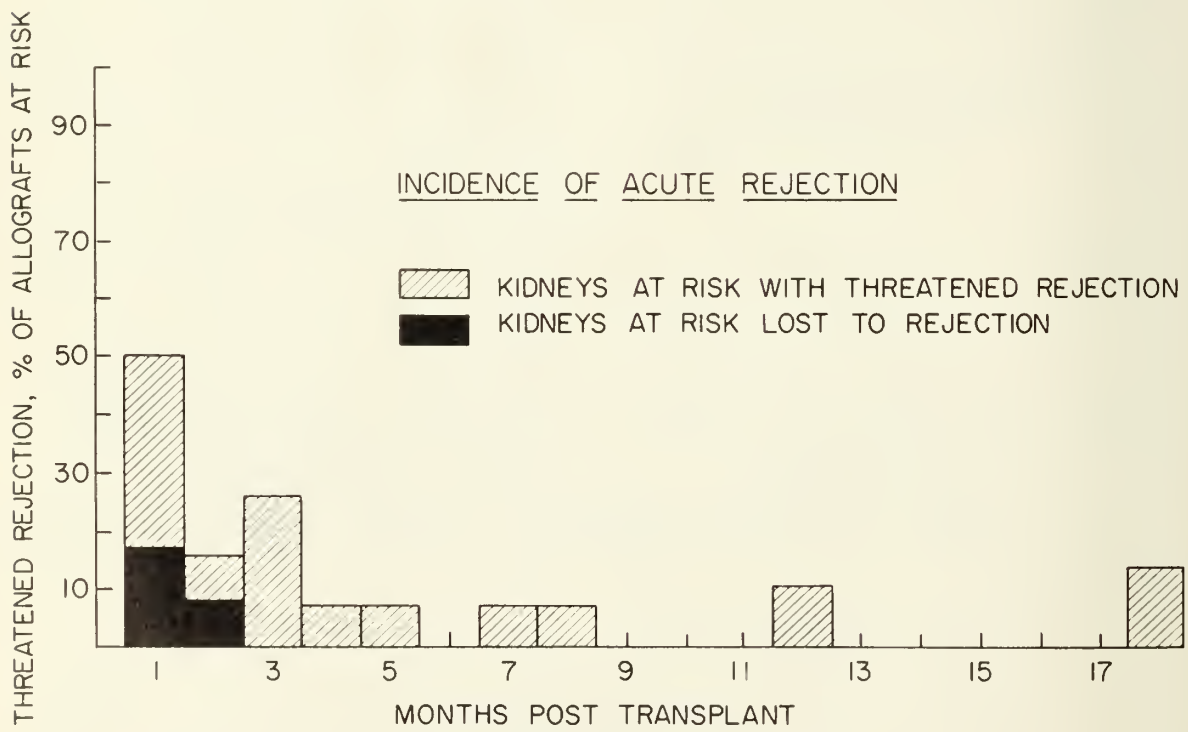

Figure 2. Incidence of Acute Rejection.

TABLE IV

KIDNEY THREATENING COMPLICATIONS

Case No.	*Interval Post Transplant*	*Problem*	*Outcome*
		Mechanical	
Cadaver 1	10 weeks	Ureteral perforation, peritonitis	Success
Cadaver 4	2 days	Perinephric hematoma	Success
Cadaver 19	3 days	Ureteral clot, obstructive nephropathy	Success
L.R. 5	3 days	Ureteral perforation, lymphocoele obstructing ureter	Success
		Allograft Retrieval	
Cadaver 2	1 day	Acute tubular necrosis	Failure
Cadaver 8	1 day	ATN	Failure
Cadaver 18	1 day	ATN	Failure
Cadaver 10	1 day	ATN	Success
Cadaver 7	2 days	ATN	Success
Cadaver 14	2 days	ATN	Success
Cadaver 15	2 days	ATN	Success

nine concentration, malaise and a low urinary sodium concentration are our most reliable signs of acute rejection.[2]

In *Figure 2,* we have displayed the temporal pattern of acute rejection episodes for both related and cadaver donor kidneys. In the first month, half of the transplanted kidneys had an acute rejection episode. Sixty-four per cent of these episodes were managed successfully. In the second month, fewer than 20 per cent of the kidneys experienced threatened rejection and fewer than 10 per cent were lost. After the fourth month the threat of rejection was quite low. One must be constantly on guard, however, since by relatively frequent observation of the patients we were able to detect, and combat successfully, acute rejections at the 12th and 18th month.

During the early months after the transplant, the transplanted kidney may also be threatened by complications other than acute rejection. These complications fall into two general areas, as shown in *Table IV.* The group of problems to which we refer as mechanical difficulties included ureteral perforation, perinephric hematoma, and ureteral obstruction. Each of these complications was handled successfully by operative intervention and none of the kidneys was lost. The other group of kidney threatening complications is related to the retrieval of the kidney from the donor. Prolonged ischemia time or other delays during organ retrieval has resulted in acute tubular necrosis in seven of the cases. Three of these kidneys never functioned; on the other hand, four of the patients were sustained with hemodialysis during the oliguric phase of acute tubular necrosis until the transplanted kidney recovered life-sustaining renal function.

The recipient of a renal transplant faces complications in addition to those which threaten the function of the allograft.[6] The immunosuppressive therapy needed to protect the kidney from rejection also makes the patient more vulnerable to infection.[7] In *Table V* we have listed some of the life-threatening infections and gastrointestinal catastrophes that we

TABLE V

LIFE THREATENING COMPLICATIONS

Case No.	*Interval Post Transplant*	*Problem*	*Outcome*
Cadaver 1	5 weeks	CMV pneumonia, bleeding ulcer	Success
Cadaver 5	22 months	Meningiococcal pneumonia	Success
Cadaver 9	6 weeks	Fungal brain abscess, rejection	Died
Cadaver 12	6 days	Perforated gastric ulcer, Bacteroides sepsis, peritonitis, rejection	Died
L.R. 4	2 months	GI bleeding	Success
L.R. 8	1 day	Anesthetic complication	Success

TABLE VI

INFECTIONS, NON-LIFE THREATENING

Urinary tract	
Bacterial	24
Fungal	1
CMV	2
Skin	
Wound	4
Herpes	5
Fungus	5
Pulmonary	
Bacterial	3
CMV	2
HAA Hepatitis	2
Epididymitis	2

have seen. Cytomegalovirus pneumonia, meningococcal pneumonia, and fungal abscesses were infections found in these patients. Three patients also had bleeding gastric ulcers, two of which were treated successfully with operation. Death occurred in two patients who had had only minimal rehabilitation from dialysis and were offered transplantation in an heroic attempt to save their lives. However, both experienced acute rejection of the allograft and died of complications related to the immunosuppressive therapy. Another patient had prolonged respiratory arrest immediately after the transplant, which was due to extreme sensitivity to the muscle relaxing agent used in the course of surgery. Most of the foregoing complications occurred in the early months post-transplantation. Once the patients passed beyond this early period, the incidence of life-threatening complications was minimal.

There were also a few minor irritating complications, as shown in *Table VI*. Urinary tract infection was a common problem which was expected in view of the recent surgery on the lower genitourinary tract. Skin infections were those expected in patients treated with high dose corticoids. There was pulmonary infection with hemophilus influenza and cytomegalovirus in two patients. There were two cases of Australian antigen hepatitis and two cases of epididymitis. This group of infections clustered temporally in the early months post-transplant.

Relatively minor non-infective complications are shown in *Table VII*. Most of the patients developed Cushingoid features in the postoperative period. Hypercalcemia due to secondary hyperparathyroidism was successfully controlled by the oral administration of aluminum phosphate gel.[8] Hypertension and obesity were usually seen together in the same individual and responded to weight reduction and reduction of corticoid dosage. We have seen a single case of aseptic necrosis of the femoral head.

TABLE VII

NON-INFECTIOUS PROBLEMS

Cushingoid features	20
Hypercalcemia	6
Cataract	4
Hypertension	4
Obesity	4
Depression	2
Aseptic necrosis, femur head	1
Carpal tunnel syndrome	1
Acne	1
Gastrointestinal bleed, mild	1

Although several of the problems we have just discussed were formidable, nearly all have been handled successfully and this group of patients has enjoyed a high level of rehabilitation. Sixteen of the patients have returned to their level of activity antedating the onset of the symptoms of renal disease. One patient is hampered due to psychological, but not kidney problems. Eight of the heads of households are working full time and paying taxes, and productively contributing to their communities in other ways. Six of the women are at full function in their homes and one student has returned to school.

In summary, we face a number of problems in the delivery of definitive renal care through the use of renal transplanation. Most of the problems can be dealt with successfully and a high degree of rehabilitation is to be expected with a renal allograft.

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Practical Coronary Surgery

Its Place in the Community Hospital

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William Heberden first described angina pectoris in 1768, and it has been a subject of deep and abiding interest to internists.[1] The events of the last decade have intensified that interest and have greatly expanded our knowledge in the field of coronary disease.

Framingham[2] and other studies have helped to identify patients at risk. Hughes Day's[3] concept of coronary care, Sones[4] contribution to angiography, and the work of Levy and Fredrickson[5] with blood lipids are but a few examples of the advances which have written a new and exciting chapter in the history of this old disorder.

In spite of all this, coronary artery disease remains the number one killer in America today. It kills over 600,000 persons each year, while millions more suffer from angina or myocardial infarction. Internists can do a lot for these patients. They can give them advice about prevention and they can help them to live more comfortably. Better care can be taken of the acute infarction, however, the progress of the disease cannot be arrested or reversed. Nor can the coronary blood flow be increased to any significant extent by medical means. Until this is achieved, probably not much headway against the mortality or the more severe forms of morbidity can be made.

The introduction of saphenous vein graft techniques by Favalaro and others,[6, 7] in 1967, has added another dimension to the treatment of angina. Here, at last, was a procedure that could offer an immediate increase in coronary flow. By 1970, several centers were reporting large series in the literature *(Table I)*.[7-11]

From these reports, it was clear that the procedure could be done in large medical centers with a mortality rate in the 5-15 per cent range. Later reports from these centers indicate an even lower rate. There is very little information regarding long-term results. The operation does nothing to stop the process of atherosclerosis and, therefore, must be considered palliative. Nevertheless, the short-term results appear to be impressive.

Since the development of the coronary care concept in the early 1960s, Bethany Medical Center, a 300-bed voluntary hospital, has seen a relatively large volume of patients with coronary disease. Our own experiences suggested that the short-term benefits alone might justify saphenous vein graft surgery if it could be performed safely in a community hospital. This report describes results in our first 100 consecutive cases (two of which were re-operations).

Methods

Patients referred by attending physicians for study received the usual comprehensive history, physical examination, EKG, chest x-ray, and appropriate chemistries. Suitable candidates were then studied by multiple injection, multiple view selective coronary cineangiography, and left ventriculography. Of 281

TABLE I
MORTALITY

Center Reporting	*Year*	*No. Cases*	*Operative Mortality %*
Milwaukee (Johnson, *et al.*)	1969	245	14
Cleveland (Favaloro, *et al.*)	1970	570	5.4
New York (Spencer, *et al.*)	1970	195	10
Houston (Methodist Hospital) (Morris, *et al.*)	1970	161	8.7
Dallas (St. Paul Hospital) (Mitchell, *et al.*)	1970	128	11.7
Kansas City, Kansas (Bethany Medical Center)	1973	100	0

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patients so studied from June 9, 1971 to November 1, 1972, approximately 25 per cent were normal. Of the remainder, 100 cases were selected for surgery. The sex and age distribution is shown in *Table II*.

TABLE II

SEX AND AGE DISTRIBUTION

	35-40	*41-50*	*51-60*	*61-69*	*Total*
Males	1	35	33	14	83*
Females	1	5	5	4	15
	2	40	38	18	98

* 100 consecutive procedures included two cases in this group who were operated twice.

Table III describes the criteria for clinical classification. Spodick[12] has warned of the danger of "numerators" without proper "denominators." We have, therefore, used a classification which considers the history, EKG, and angio findings while permitting easy application in a clinical setting.

A standardized preoperative regimen was begun on elective cases at least 36 hours prior to operation. Cardiopulmonary bypass, using a Travenol bubble oxygenator, was managed by a permanent operating room pump team. A nurse from the intensive care unit (ICU) was assigned to intercept the patient coming out of surgery and to supervise recovery. Patients were then taken to ICU where the EKG, mean arterial pressure, central venous pressure, mean left atrial pressure, arterial blood gases, serum K+ levels, chest tube drainage, and urine output were monitored. Endotracheal tubes were removed on the morning after surgery. Chest tubes were removed on the third day. Patients were usually able to leave the ICU on the third or fourth day.

Table IV summarizes the procedures performed and their distribution according to clinical classification. There was almost equal distribution between Groups I, II, and III. Tables V, VI and VII provide data concerning hospital stay and pump time for various types of single, double, and triple grafts.

TABLE III

DIAGNOSTIC CLASSIFICATION

Group I
- A) Angina.
- B) No myocardial infarction.
- C) High grade or complete segmental obstruction in the proximal portion of one or more major coronary branches.
- D) No significant diffuse distal disease.
- E) Good distal run-off.
- F) Good left ventricular contraction.
- G) Normal LVEDP.

Group II
- A) Angina.
- B) May have had previous infarction.
- C) Segmental proximal obstruction.
- D) Some diffuse distal disease but good run-off.
- E) Good left ventricular contraction.
- F) LVEDP less than 15 mm Hg.

Group III
- A) Congestive heart failure.
- B) May also have angina or arrhythmias.
- C) Previous infarction—usually massive or multiple.
- D) Occlusion of one or more major vessels.
- E) Significant distal disease.
- F) Poor left ventricular contraction.
- G) LVEDP greater than 15 mm Hg.

Group IIIa
- Same as above but with resectable left ventricular aneurysm.

Group IIIv
- Same as above but candidate for aortic or mitral valve replacement or repair.

Group IV
- Emergency surgery
  - A) Coronary insufficiency—impending infarction.
  - B) Acute infarction.
  - C) Arrhythmias.

Complications

The usual minor complications were encountered. These included transient hypotension, oliguria, anemia, atelectasis, and the post-thoracotomy syndrome. These were not life threatening and were usually resolved well before discharge.

Two patients of Group I required re-operation.

Serious complications are listed in *Table VIII*. Some patients had more than one, therefore, the number of patients is smaller than the number of complications. It should be noted, in contrast to the literature, that there were no instances of severe bleeding, no transfusion reactions, no renal shutdown, and no serious problems related to cardiopulmonary bypass. There were no serious infections. Only one serious arrhythmia was encountered early. One patient later required an elective pacemaker insertion.

Graft closure was found in two Group I patients who were re-studied because of recurrent symptoms. One patient was re-operated at ten months and the other at eight months. Both are now asymptomatic.

Deaths

There was no operative mortality. In followup from June 9, 1971 to February 1, 1973 (20 months),

TABLE IV
PROCEDURES

Group	*Sex*	*Av. Age*	*Single Grafts*	*Double Grafts*	*Triple Grafts*	*Aneurysm Resection*	*Valve*
I	23M	51	15 (a)	12	1		
	5F	52					
II	29M	54	6	19	10		
	6F	52					
III	23M	55	5	8	11		
	1F	61					
IIIa	6M	49	2	4	1	7	
	1F	50					
IIIv	2M	50	0	3	0		3
	1F	51					
IV	2M	54	1(b)	1 (c)		2	
	1F	51		1 (d)			
Total	100		29	48	23	9	3
Total grafts ..	194		29	96	69		

(a) Includes two patients re-operated because of graft closure.
(b) Single graft + aneurysm resection.
(c) Double graft + aneurysm resection.
(d) Double graft only.

there were five late deaths (approximately 3% per year). Amsterdam[13] reported 16 deaths in a series of 66 cases studied by angiography and followed on medical management for 26 months (12% per year). Moberg[14] reviewed 200 cases followed medically for seven years and found an annual mortality rate of 4 per cent for anterior descending disease, 1.8 per cent for single right or circumflex lesions. However, patients with 2- and 3-vessel disease had an annual rate of 6.3 and 10 per cent respectively. Furthermore, patients with poor left ventricular function were excluded from his study.

These are not conclusive arguments, but they lead us to consider our late mortality rate favorable for the short-range followup. *Table IX* outlines the findings in our patients who succumbed. Three were Group III poor-risk candidates with poor left ventricular function. Of these, death was due to congestive heart failure in two and to graft closure in one. One Group I patient died because of graft closure due to kinking by adhesions. One Group II patient

TABLE V
SINGLE VESSEL GRAFTS

Vessel	*No.*	*Av. Days From Surgery to Discharge*	*Av. Total Pump Time in Minutes*
RCA	12 (a)	11.7	36.3 (c)
LAD	13	9.6	30.9
LAD:DIAG	1	85. (b)	20.0
CX	3	11.0	49.0
	29	10.6	36.0

(a) Includes 3 patients who also had aneurysm resection.
(b) Excluded from average.
(c) Includes 2 patients done without pump.
Abbreviations: RCA = Right Coronary Artery; LAD = Left Anterior Descending; LAD:DIAG = Major Diagonal Branch of LAD; CX = Left Circumflex Artery.

TABLE VI
TWO-VESSEL GRAFTS

Vessels	*No.*	*Av. Days From Surgery to Discharge*	*Av. Total Pump Time in Minutes*
RCA-LAD	25 (a)	10.4	53.0
RCA-CX	10 (b)	14.3	65.5
LAD-CX	8 (a) (c)	11.6	85.2
LAD-LAD:DIAG	5 (a)	14.8	52.2
	48	11.9	61.1

(a) Includes one patient who also had aneurysm resection.
(b) Includes two patients who also had aneurysm resection.
(c) Includes three patients who also had valve replacement or repair.

TABLE VII

THREE-VESSEL GRAFTS

Vessels	*No.*	*Av. Days From Surgery to Discharge*	*Av. Total Pump Time in Minutes*
RCA-LAD-CX	15 (a)	13.5	74.5
RCA-LAD-LAD:DIAG	5	17.2 (b)	68.6
RCA-LAD:DIAG-CX	1	18.0	56.0
LAD-LAD:DIAG-CX	2	13.0	72.0
	23	14.4	72.1

(a) Includes one patient who also had aneurysm resection.
(b) Includes one patient whose stay was 45 days.

(For Tables IX, X, and XI see following pages.)

died because of occlusion due to progression of disease in the ungrafted RCA. Grafts into the LAD and into the LAD:DIAG were open and functioning. This patient had a previous anterior wall myocardial infarction.

Results

Much thought was given to the methods to be used in evaluating our results. It was obvious from the beginning that systematic angiographic re-study, as well as pre and postoperative stress testing, were not possible in our setting. Many authors[12, 15-17] have pointed out the fallacy of subjective improvement alone as a measure of results. In fact, it may prove difficult to accurately assess the effects of this operation by any means. Nevertheless, we are determined to follow our patients as carefully as possible, and to draw only those conclusions which are justified by the data.

TABLE VIII

COMPLICATIONS

EARLY (During the surgical hospital stay)	
Myocardial infarction	7
Stroke	3
Brain damage due to hypoxia	2
Transient hemiparesis	1
Psychosis	1
Congestive heart failure	7
Arrhythmia (Stokes-Adams)	1
Atelectasis (bronchoscopy)	1
Severe bronchospasm	2
Sternal wound dehiscence	1
LATE (Requiring readmission)	
Myocardial infarction	3
Arrhythmia (pacemaker insertion)	1
Osteomyelitis of sternum	1

Table X is a questionnaire completed by the attending physician for each patient at six-month intervals. These findings, along with our own observations, are correlated with the clinical classification in *Table XI*. During the follow-up period of 3 to 20 months, 51 per cent of the patients were asymptomatic. Another 34 per cent were improved. Only 15 per cent were no better, were worse, or dead. It is interesting to note that the results in Groups I, II, and III were similar despite the gross dissimilarity in disease. The patients in Group II who were worse all had neurologic rather than cardiac disability.

Summary

Between June 9, 1971 and November 1, 1972, 194 reversed saphenous vein aorto-coronary bypass grafts were performed in 100 consecutive operations on 98 patients, with followup to February 1, 1973. There were no operative deaths. There were five late deaths. A comparison with the literature is made in *Table I*. Short-term results appear to be favorable. Long-term results remain to be evaluated.

Acknowledgment

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TABLE IX

DEATHS

Pt. No.	Group	Angio Findings	Date of Surgery	Procedure	Hosp. Stay Days	Pump Time Min.	Post-op Complications	Date of Death	Autopsy Findings
28	IIIv	80%—LAD TB-LAD:DIAG TB-CX Mitral insuf. Poor contrac.	11-30-71	LAD CX Mitral valve Anuloplasty	14	98	CHF	12-15-72	
31	III	90%—RCA 85%—LAD TB-LAD:DIAG Poor contrac. LVEDP = 0-24	1-3-72	RCA LAD LAD:DIAG	11	78		DOA 5-12-72	Stenotic closure of aortic ostia of LAD + LAD:DIAG grafts
37	IIIa	TB-RCA 85%—LAD 90%—CX Aneurysm Poor contrac. LVEDP = 0-40	1-26-72	RCA CX Aneurysm Resection	37	113	CHF Stokes-Adams	DOA 3-21-72	
54	I	95%—LAD Good contrac. LVEDP = 0-12	3-15-72	LAD	9	23		DOA 6-8-72	Graft kinked by adhesions
61	II	35%—RCA 85%—LAD Good contrac. LVEDP = 0-12	4-5-72	LAD LAD:DIAG	14	63		DOA 11-29-72	Grafts open RCA occluded by new disease

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TABLE X

BETHANY MEDICAL CENTER
CARDIOVASCULAR DEPARTMENT

Dear Doctor:

We are interested in the long term follow-up of patients who have had coronary saphenous vein graft surgery at Bethany Medical Center. We are asking you to help us objectively evaluate the effectiveness of this procedure by answering the following questions about your patient.

NAME ..... DATE .....

1. The patient is Living ....
   Dead .... Date of Death ....
2. Since surgery, has the patient had any cardiac problems requiring hospitalization? Yes ....
   No ....
   Please explain .....
   .....
   .....
3. Surgery was performed because of:
   Angina ....
   Congestive failure ....
   Impending infarction ....
   Arrhythmia ....
   Other .....
4. Does the *patient* think he/she is:
   Considerably better ....
   Some better ....
   No better ....
5. Do *you* think the patient is:
   Considerably better ....
   Some better ....
   No better ....
6. What symptoms does the patient have now:
   None ....
   Angina ....
   Dyspnea ....
   Arrhythmia ....
   Other .....
7. If it were necessary, do you think that the patient would be willing to have the operation again?
   Yes ....
   No ....
8. Do you think the patient would be willing to have a coronary artery restudy?
   Yes ....
   No ....
9. Please give us your comments on the back of this sheet.

We will plan to ask you these, or similar questions at six month intervals. If there is information you would like to give us in the interim, please do not hesitate to do so. Thank you again for your help in this evaluation.

Franklin G. Bichlmeier, M.D.
Sherman M. Steinzeig, M.D.
Thomas V. Thomas, M.D.
William B. Toalson, M.D.

Signed ..... M.D. Date.....

Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high-contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

Practical Coronary Surgery

(Continued)

TABLE XI

CLINICAL RESULTS

Group	*No.*	*Asymptomatic*	*Considerably Better*	*Some Better*	*No Better*	*Worse*	*Late Deaths*
I	28	17 (61%)	2 (7%)	5 (18%)	3 (11%)		1 (3%)
II	35	17 (49%)	6 (17%)	6 (17%)	1 (3%)	4 (11%)	1 (3%)
III	24	14 (58%)	4 (17%)	3 (13%)	2 (8%)		1 (4%)
IIIa	7	1 (14%)	2 (29%)	3 (43%)			1 (14%)
IIIv	3	1 (33%)	1 (33%)				1 (33%)
IV	3	1 (33%)	1 (33%)	1 (33%)			
Total	100	51	16	18	6	4	5

The Fall Meeting

of the

KMS HOUSE OF DELEGATES

Sunday, November 18, 1973–10:00 A.M.

University Ramada Inn, Manhattan

One-day business meeting to consider items relating to legislation and others that cannot be delayed until May. It is hoped all component societies will be represented by their full complement of delegates.

All members of the Society are invited.

ALL RESOLUTIONS MUST BE SUBMITTED NOT LATER THAN NOVEMBER 1, 1973. THEY WILL BE REPRODUCED IN SUFFICIENT QUANTITY TO PASS OUT TO THE DELEGATES AT THE MEETING.

Persistent Ventricular Tachycardia

The Use of Diphenylhydantoin

CHIH-PING YANG, M.D., ***Topeka***

WHEN FACED with an intractable ventricular tachycardia and bewildered by the failure of treatment, try Diphenylhydantoin (DPH);[1, 2] it could be life saving. DPH is effective in ventricular tachycardia due to circus reentry[3, 4] because it improves conductivity of cardiac tissue and decreases unidirectional block. DPH also, by suppressing Phase 4 depolarization, is effective in treating ventricular tachycardia due to enhanced automaticity.

Case Report

A 56-year-old male was admitted to our hospital on July 10, 1972, with epigastric pain, precordial chest pain radiating to the neck and to the left shoulder associated with diaphoresis, and general weakness. The patient had a previous history of hiatus hernia, peptic ulcer, and arterial hypertension. He had smoked two packs of cigarettes a day for years. His blood pressure was 160/90 mm Hg, with early hypertensive retinopathy. The neck veins were flat. There were a few moist rales bilaterally. Auscultation revealed a regular rhythm of 92 beats per minute, S_1 and S_2 normal, intermittent S_3, prominent S_4, and no murmurs. Rectal examination revealed no tarry stool.[5] EKG revealed acute transmural inferior and lateral wall myocardial infarction, right bundle branch block, and left anterior hemiblock *(Figure 1)*.[6] Serum glutamic-oxaloacetic transaminase (SGOT), lactic acid dehydrogenase, and creatine phosphokinase were markedly elevated (SGOT, 330 units). Procainamide, 500 mg orally, was immediately given for antiarrhythmic prophylaxis;[7, 8] and was repeated every four hours. Six hours after admission, the patient developed pulmonary congestion, S_3 gallop, and grade II/VI apical holosystolic murmur compatible with papillary muscle dysfunction.[9] The neck veins became distended and the central venous pressure (CVP) was 15 cm H_2O.[10] Digoxin, 0.5 mg orally, was then given. This medication was continued at 0.25 mg twice a day. Second-degree A-V block with Wenckebach's phenomenon developed after a total dose of digoxin (0.75 mg), but converted to regular sinus rhythm with the intravenous dose of 0.6 mg Atropine Sulfate *(Figure 2)*.[11, 12] Lasix and Dyrenium were also given for left ventricular failure. Serum electrolytes were normal. There was persistent chest pain (despite the frequent administration of narcotics), sustained fever, leukocytosis, high SGOT and left ventricular failure, all indicating poor prognosis. On the second day, a pericardial friction rub was noted. Anticoagulant had not been used. The patient improved but remained critical. On the tenth day, ventricular premature beats developed; procainamide was discontinued, and intermittent Lidocaine drip was given intravenously, with good response.[13] Also administered was a dose of Valium, 5 mg orally, four times a day. On the 13th day, profound heart failure was improved by booster doses of Cedilanid, totaling 0.6 mg. Ventricular fibrillation developed on the 14th day, and was converted to sinus rhythm with 400 watts/second DC defibrillation *(Figure 3)*, but ventricular tachycardia recurred and persisted *(Figure 4)*. There was no response to two 100-mg lidocaine boluses, or to eight applications of DC countershock treatments. Further lidocaine doses, totaling 1 gm, converted the ventricular tachycardia into ventricular trigeminy; how-

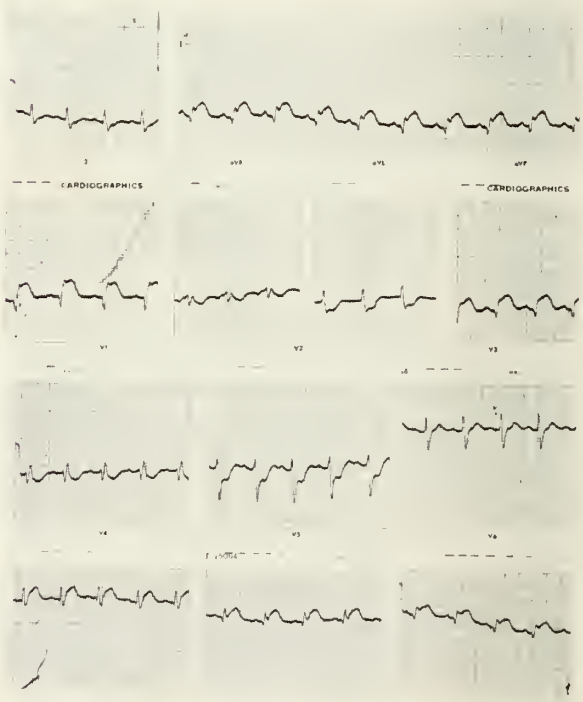

Figure 1

From the Department of Medicine, Veterans Administration Hospital, Topeka, Kansas 66622.

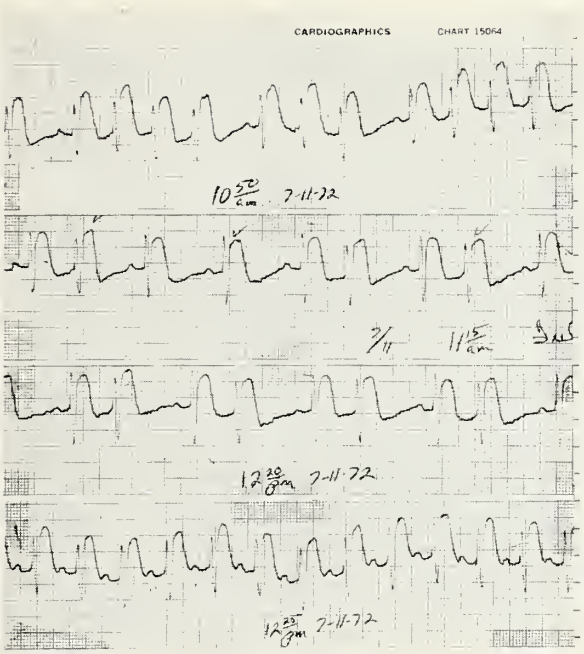

Figure 2

ever, ventricular tachycardia recurred immediately. The patient developed convulsions,[14] which subsided after a dose of 15 mg Valium given intravenously. Four more attempts at DC countershock failed to convert the ventricular tachycardia. Following 250

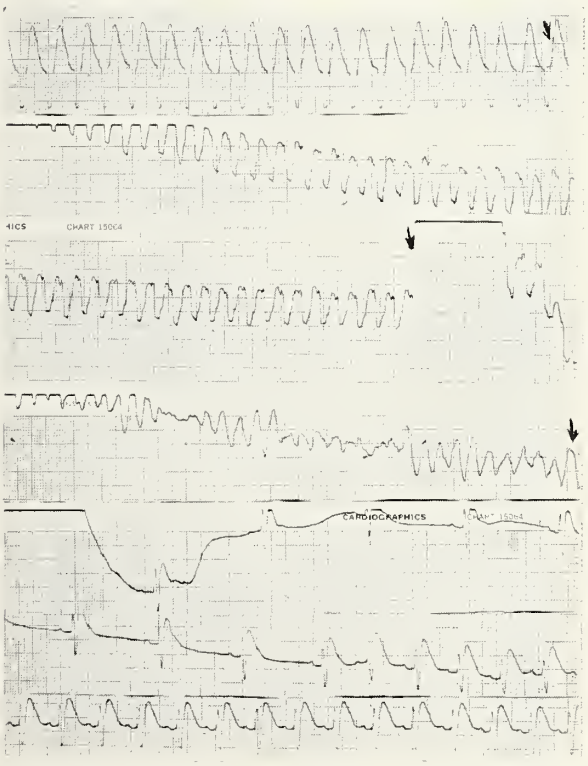

Figure 3

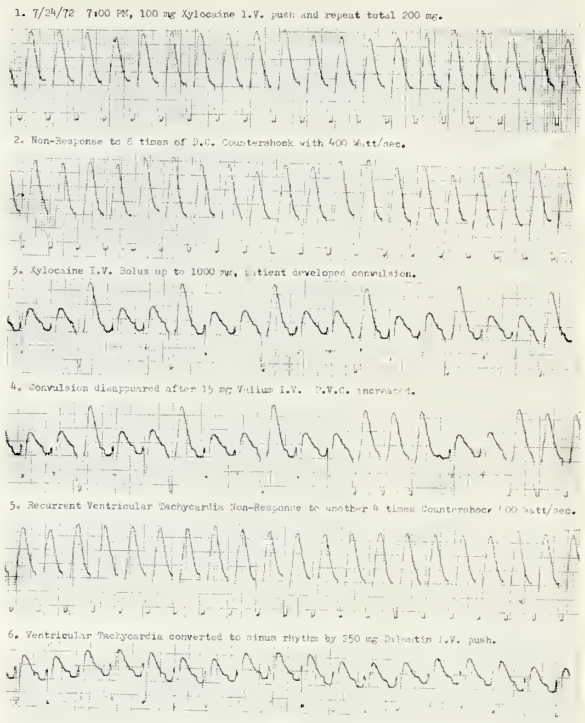

Figure 4

mg of DPH, administered intravenously, it was converted to sinus rhythm, which remained only for 20 minutes. The patient again developed ventricular trigeminy, with recurrent ventricular tachycardia *(Figure 5)*, which did not respond to treatment with propranolol (10 mg intravenously). Again, a dose of 250 mg DPH intravenously converted the ventricular tachycardia to sinus rhythm. Ventricular tachycardia with re-entry phenomenon *(Figure 6)* recurred the following morning, and was for the third

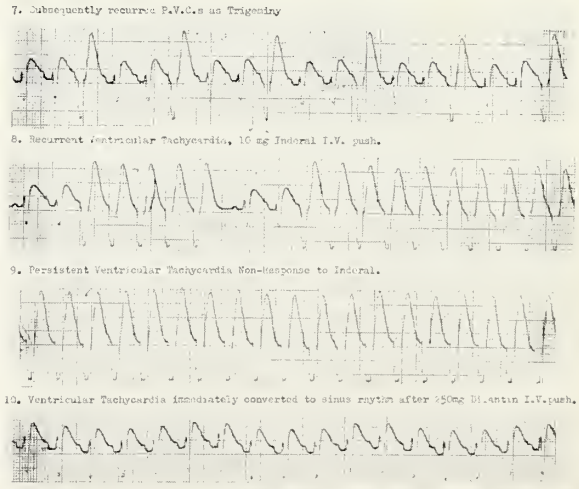

Figure 5

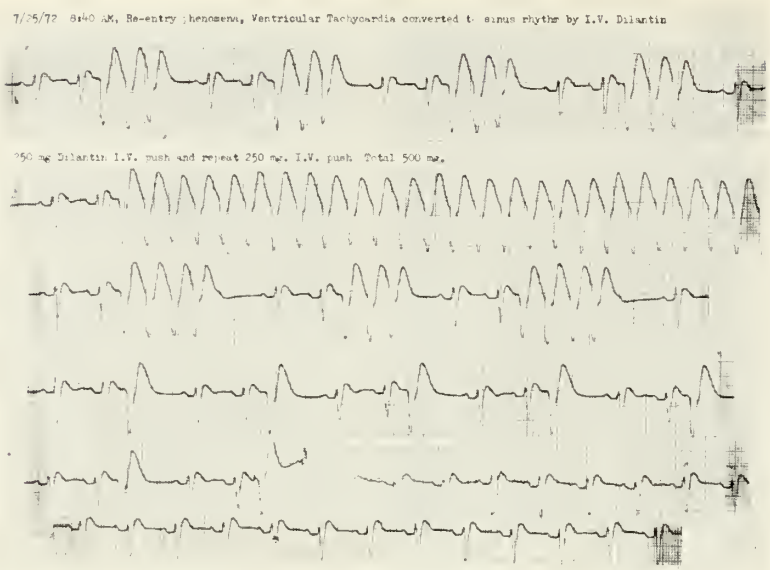

Figure 6

time converted to sinus rhythm with two 250-mg boluses of DPH, administered intravenously. Oral maintenance with 100 mg DPH was continued every four hours; daily DPH serum level ranged from 6 Ug/ml to 13 Ug/ml. The patient remained in normal sinus rhythm, and oral doses of 0.25 mg digoxin twice a day were continued.

Discussion

This is a case of intractable ventricular tachycardia following acute myocardial infarction. Procainamide, large doses of lidocaine, repeated DC countershock, and propranolol failed to convert this life threatening cardiac arrhythmia which was so dramatically controlled with DPH. DPH was credited with abolishing the persistent ventricular tachycardia, which was believed to be due to circus re-entry rather than digitalis intoxication; it also permitted continuous digitalization when digitalis was so critically needed; yet, the therapeutic and toxic doses were very close. DPH may transiently depress left ventricular function,[17] but the inotropic action of digitalis can combat this depression. DPH has been reported effective in ventricular tachycardia following cardioversion or anesthesia; it was also observed that DPH had generally good effect in digitalis-induced arrhythmia, but supplement potassium and correction of electrolyte and acid-base imbalance could be more rewarding in suppression of a digitalis-induced repetitive ventricular tachycardia.

There was no electrolyte or acid-base imbalance; the arterial blood gas revealed no hypoxemia in our patient receiving 40 per cent oxygen. Although DPH has multiple pharmacologic actions, its action on the re-entry phenomena is unique, and even in low concentration can be effective in terminating the re-entry mechanism by improving conduction. The adequate dose of DPH has been variously suggested: Rosen, Lisack, and Rubin gave 5 mg/kg body weight intravenously, not exceeding 350 mg in any patient.[18] Friedberg stated that 5 to 20 mg/kg body weight may be given intravenously in a 15-minute period if the dose is controlled by monitoring the cardiac rhythm.[19] Mercer and Osburne suggested a single dose approach, to be accomplished in the following manner: an initial bolus of 125 to 250 mg, not to exceed 250 mg in 15 minutes, or 500 mg in an hour.[1] The maximum daily dose has not been established and since DPH is conjugated in the liver, the liver function of each individual should be taken into consideration.

Several deaths after intravenous administration of DPH have been reported,[20] and slow administration of DPH with simultaneous cardiac monitoring is thus very important. It is not proper to give DPH intramuscularly in life threatening arrhythmia, because absorption is delayed. Koch-Weser[8] mentioned that the usually effective plasma level of DPH for prevention of cardiac arrhythmia was from 8 to 16 Ug/ml; toxicity may occur at levels greater than 16 Ug/ml. We reached the desired effect in our patient with plasma level ranging from 6 Ug/ml to 13 Ug/ml. Once the ventricular tachycardia is controlled by intravenous administration of DPH, the oral maintenance with DPH should be started immediately, and 100 to 200 mg every six to eight hours should prove to be generally adequate.

Eight months following myocardial infarction, our patient was on a regimen of digoxin, 0.25 mg daily, administered orally, and 100 mg DPH, three times

a day, administered orally. The patient maintained a regular sinus rhythm, and there was no untoward effect after this long-term administration.[8]

Summary

A 56-year-old male developed persistent ventricular tachycardia following acute myocardial infarction. This life threatening arrhythmia did not respond to large doses of intravenous administration of lidocaine, twelve 400-watts/second DC countershock treatments, or intravenous administration of propranolol. The ventricular tachycardia in this case was dramatically converted to regular sinus rhythm with intravenous administration of DPH. Subsequent oral DPH doses maintained an adequate DPH plasma level, and a recurrence of arrhythmia was prevented. The patient tolerated this long-term program well and with no appreciable side effects.

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NEW MEMBERS

The JOURNAL *takes this opportunity to welcome these new members into the Kansas Medical Society.*

Mammo Amare, M.D.
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LEADERSHIP CONFERENCE

February 1-3, 1974

The Crown Center Hotel—Kansas City, Missouri

All members, and especially the presidents and secretaries of component county medical societies, are earnestly urged to attend this first annual conference which will include seminars and sections on administrative and organizational problems at the local level.

The speakers will include:

Malcolm C. Todd, M.D., Long Beach, Calif.—President-Elect, American Medical Association.

Jerome P. Lysaught, Ph.D., Rochester, New York—Director, National Commission for the Study of Nursing and Nursing Education.

Bill Roy, M.D., Washington, D. C.—U. S. Congressman, Kansas Second District.

Bernard P. Harrison, J.D., Chicago, Ill.—Director, Medical Practice Division, American Medical Association.

William O. Rieke, M.D., Kansas City, Kansas—Vice-Chancellor for Health Affairs, University of Kansas Medical Center.

Robert Haakinson, Ph.D., Philadelphia, Pa.—Associate Dean, School of Business Administration, Temple University.

The program will center around organizational and administrative problems encountered in local societies in communication with KMS and utilization of available information; legislative update—federal and state; the changing role of nursing in today's medicine; AMA and its relationship to the local practicing physician; and other topics.

More information will be forthcoming by mail and in subsequent issues of the JOURNAL. If you have questions, please contact the KMS Executive Office, either by calling collect, (913) 235-2383; or by writing, 1300 Topeka Avenue, Topeka, Kansas 66612.

ALL KMS MEMBERS ARE INVITED TO ATTEND

Reflux Esophagitis

A Comparison of Old and New Medical Management

A. EUGENE GROSSMANN, M.D., ARTHUR P. KLOTZ, M.D., F.A.C.P., JAMES B. RHODES, M.D. ***and*** **THOMAS KORB, M.D.,**
Kansas City, Kansas

STANDARD medical and surgical treatments for reflux esophagitis are designed to prevent reflux of gastric acid-pepsin into the esophagus.[1] A standard medical therapy includes elevation of the head of the bed six to eight inches with bed blocks, avoidance of tight abdominal garments, elimination of food four hours before retiring, weight reduction, and a bland diet. Antacid therapy is used to neutralize the gastric acid and to increase the lower esophageal sphincter (LES) pressure through the increased release of gastrin.[2] An additional mode of medical therapy has been developed recently, which employs a chewable foaming tablet that is designed to coat the distal esophagus and locally protect against acid-pepsin gastric reflux. The tablet (Gaviscon®) contains aluminum hydroxide gel, magnesium trisilicate, sodium bicarbonate, and alginic acid. This study was designed to investigate the clinical effectiveness of the foaming agent in the treatment of reflux esophagitis.

Fifty patients with symptomatic reflux esophagitis were evaluated by upper gastrointestinal x-ray, esophageal motility study and esophagoscopy, and were then treated with a standard medical regimen to which either a foaming or a non-foaming antacid was added. The patients' initial symptoms did not provide a good indication of the extent of esophagitis at esophagoscopy. Patients treated with either antacid showed subjective and objective improvement.

Methods

Fifty patients with symptoms of reflux esophagitis were evaluated. Each patient had a history, physical examination, and upper gastrointestinal x-ray before entering the study. A printed questionnaire was completed on each patient and the subjective findings were graded in four categories: (1) Epigastric to retrosternal distress; (2) Regurgitation; (3) Epigastric gas; and (4) Motor symptoms of swallowing. A grade of zero was given for no symptoms, one for mild, two for moderate, three for severe, and four for intolerable symptoms. All of the patients underwent an esophageal motility with measurement of the LES pressure and esophageal peristalsis. Esophagoscopy was performed on every patient with the Olympus EF-II fiberoptic esophagoscope, and color photographs of the distal esophagus were made. The degree of esophagitis was graded on a five-point scale: zero for none, one for erythema, two for erythema and exudate, three for erythema, exudate and friability, and four for erythema, exudate, friability, and ulceration. The patients were instructed on the standard medical program and assigned by a random table to either a foaming or a non-foaming antacid tablet. The non-foaming tablet contained a balance of magnesium and aluminum hydroxide in a well-established preparation (Maalox® Number Two). Both types of antacids were given in the same dosage—two tablets chewed one-half hour after meals and at bedtime. Evalaution of the subjective response was made at two and four weeks. The objective response was evaluated at four weeks by repeating the esophagoscopy if esophagitis was present on the initial endoscopy.

Results

None of the patients had an increase in symptom score as a result of therapy. Three of the 50 patients had incomplete evaluations—two were lost to follow-up and one refused to take Gaviscon because of nausea. Evaluation of symptom scores after four weeks of therapy is shown in *Table I*. The foaming antacid showed a statistically significant difference from the nonfoaming antacid only with respect to retrosternal and epigastric distress ($p < .05$). The differences between the two drugs in the other three symptoms, though favoring the foaming antacid, were not found to be statistically significant.

The patients were divided into four groups according to the initial total symptom score and the ini-

Presented at the annual meeting of the Kansas Chapter, American College of Physicians, Wichita, Kansas, February 23, 1973.

TABLE I

THERAPEUTIC RESPONSE TO FOAMING AND NON-FOAMING ANTACIDS

	Patients	*Excellent*	*Intermediate*	*Poor*
Foaming	25	10 (40%)	10 (40%)	5 (20%)
Non-foaming .	25	6 (24%)	8 (32%)	11 (44%)

tial grade of esophagitis *(Table II)*. Patients with little or no esophagitis at endoscopy (Groups A and C) comprised 70 per cent of the total patients. About one third of the patients with mild esophagitis on endoscopy had severe symptoms. If the patients whose esophagitis was grade 3 or 4 (Groups B and D), one third had low initial symptom scores. The patients with low initial symptom scores (Groups A and B) tended to have a mild degree of esophagitis (82%). However, in the patients with high initial symptom scores (Groups C and D) there was a nearly equal distribution of mild and severe esophagitis. The patients with severe symptoms and little esophagitis were found to have a high frequency of diffuse esophageal spasm. The history alone did not correlate well with the degree of esophagitis at endoscopy.

Twenty-three patients had esophagoscopy before and at the end of one month of treatment. The average grade of esophagitis was the same in the foaming and non-foaming antacid groups at the beginning of treatment, and there was no significant difference between the two antacid groups after one month of treatment *(Table III)*. Both groups showed objective improvement in the esophagitis.

Discussion

Symptomatic reflux esophagitis may produce numerous symptoms that may be interpreted as being due to a variety of disorders: gastric or duodenal ulcers, gallbladder disease, irritable bowel syndrome, cardiac disease, chest wall pain, and others. The patients in this study were thought to have classical symptoms of reflux esophagitis and each patient's subjective complaints were evaluated in only four categories. Consequently, there may be a number of patients with reflux esophagitis who do not have symptoms in any of these four categories but who may respond to a standard medical regimen. Moreover, the frequency of antacid administration used in the study may not be adequate to produce a therapeutic response in all patients.

TABLE II

CLINICAL GROUPS

Group	*Sum of Symptoms*	*Degree of Esophagitis*	*Number of Patients*
A	1-8	0-2	22
B	1-8	3 and 4	5
C	9-16	0-2	13
D	9-16	3 and 4	10
			—
			50

TABLE III

OBJECTIVE RESPONSE AVERAGE GRADE OF ESOPHAGITIS

	Number of Patients	*Initial*	*Four Weeks*	*Change*
Foaming	10	2.5	1.7	0.8
Non-foaming	13	2.5	1.8	0.7

Conclusion

Medical therapy for reflux esophagitis can produce both subjective and objective improvement. The history alone is not a reliable index of the degree of esophagitis. The addition of a foaming antacid to a standard medical program for reflux esophagitis may be beneficial in patients with retrosternal or epigastric distress.

References

1. Skinner, D. B.; Belsey, R. H. R.; Hendrix, T. R. and Zuidema, G. D.: *Gastroesophageal Reflux and Hiatal Hernia.* Boston, Little Brown and Co., 1972, p. 133.

2. Castell, D. O. and Harris, L. D.: Hormonal control of gastroesophageal sphincter strength. *N. Engl. J. Med.* 282:866, 1970.

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

The President's Message

The physicians in the state of Kansas have traditionally had excellent rapport with the political system in the state. We have attempted over a very long period of time to participate in this political mechanism without much regard as to the deleterious effects it may have on an individual physician. We have placed the good of the patient primarily in our discussions throughout all these years. We have consistently felt that the interest of the patients should come first and that the interest of the physician should come secondarily. This, I think, has been the atmosphere under which we have worked over a long period of time. We have lost some of our battles, we have won most of them, and I think this is the way it ought to be. Because part of this process is selling, we can attempt to instruct our political representatives in an unbiased manner, and when failing, then the failure may be ours in not properly orienting these people to the problem and its proper resolution. On the other hand, it appears to me that the participating physicians have had little input into many of our representatives at the federal level.

I feel that part of the answer may lie in the following: people running for a federal job in Congress need money. It is a well known truism that physicians do not contribute money. We know, however, that the cultist members of our society contribute great amounts of money per individual at all levels of our political process. The time for contributions to your political candidates is rapidly approaching and I would encourage each of you to look deeply into your own feelings about this.

We really can get something for nothing—whether you like or not.

Thomas F. Taylor

President

For the Record

By-Laws, Kansas Medical Society:
10.0 THE EDITORIAL BOARD

10.1 *Composition and Appointment*
The Editorial Board is comprised of five (5) or more members, appointed for terms of three (3) years by the Council; no more than two (2) appointments for a complete term are made in a single year. When a vacancy occurs it is to be filled for the unexpired term at the next meeting of the Council.

10.2 *Duties of the Chairman*

10.21 To direct the operation of the Board.

10.22 To be the Editor of the JOURNAL OF THE KANSAS MEDICAL SOCIETY.

10.23 To make an annual report to the House of the activities of the Board and the status of the JOURNAL.

10.3 *Duties of the Board*

10.31 To edit, compile, publish and distribute all publications of this Society and make business arrangements for publication as authorized by the House of Delegates or the Council.

10.32 The Board may appoint associate editors for the JOURNAL from any or all Council districts. Associate editors will assist the Board in securing information for publication, in forwarding suggestions for improvement of the JOURNAL, and supplementing the business functions associated with publication.

10.33 The Board with its associate editors will meet each year at the annual session and at the call of the chairman.

10.34 The Board collects and records data on deaths of Society members and other non-member physicians in the state. Data is published as it is received and a compilation of the previous year's deaths is reported at the annual session.

10.4 *Financial Regulations*

10.41 The Board is enjoined to maintain the JOURNAL as nearly self-supporting as possible, but as the official publication of the Society, it deserves the financial assistance necessary to correct a reasonable deficit. If a deficit exceeds budgeted money, a financial report is sent to the Council, which may appropriate additional funds.

10.42 Accounting procedures of the JOURNAL and other publications will be kept in separate ledgers and its accounts shall be maintained apart from other Society funds.

10.43 Expenditures authorized by the Board are paid by vouchers signed by the chairman and countersigned by the managing editor or the business manager of the JOURNAL. The three signatories will be individually bonded for a sum not less than five thousand dollars ($5,000). Certification of vouchers is submitted to the treasurer of the Society monthly.

10.44 Surplus funds accruing to the JOURNAL or other publications at the end of the fiscal year are deposited to a reserve account within limits established by the House of Delegates or the Council. Surplus funds beyond that limit will revert to the general fund of the Society.

The foregoing extract from the By-Laws of the Kansas Medical Society defines the official relationship of the JOURNAL OF THE KANSAS MEDICAL SOCIETY to its parent organization.

Historically, the JOURNAL is the outgrowth of the Transactions of the Society, which were the recorded papers and activities originating at the meetings of the Society from its inception. In 1901, the Society, growing with the state, recognized the desirability of having an official means of communication for the membership. The record indicates that the Founding Fathers were apparently more concerned about promoting more subscribers and obtaining the most favorable postal rates than the content of the JOURNAL. The absence of recorded evidence that the Council has entered into the actual functioning of the Editorial Board may be taken as indication that Section 10 is at least as important for what it does not say as for what it does.

On the basis of its constitutional directive, the Editorial Board has operated on the principle that its obligation and responsibility to the Society was to provide a journal which would serve the profession in Kansas with a repository for scientific articles, a source of information of Society activities, an official record of Society business, and a means of com-

munication of relevant ideas and opinions among the individual members.

The Editorial Board deems it advisable, in the absence of any previous statement, to establish certain principles and policies for the record.

Content

The following material is deemed suitable for inclusion in the JOURNAL OF THE KANSAS MEDICAL SOCIETY.

1. Scientific articles suitable for the JOURNAL are accepted from within the state and from outside sources as well, with preference given to in-state sources if the occasion for choice arises. Scientific material to be considered is expected to represent legitimate, ethical exposition of medical or medically-related subjects.

The Board considers scientific texts to be the product and responsibility of the authors. The responsibility of the Board rests in its function of evaluating and editing such material for publication, or rejection if the material is not deemed suitable. The Board seeks to maintain the highest possible professional quality in the material accepted but does not consider personal differences of opinion or a controversial subject to be a valid cause for rejection unless a majority of the Board determine them to be detrimental to the JOURNAL or to the Society.

While primary authorship is ordinarily limited to Doctors of Medicine or recognized health professionals, this does not preclude consideration of papers from other legitimate sources.

2. Medically related, non-scientific articles such as papers relating to the socio-economic aspects of medicine are considered desirable as a means of bringing before the readership information and interpretation of activities in these areas. The controversial nature which may be inherent in such articles is not considered a valid basis for rejection if the presentation of the material serves a desirable purpose in presenting alternative views and options and promoting informed debate within the Society regarding such matters. The printing of such material, therefore, is not to be construed as official opinion of the Board or the Society unless so stated, nor any commitment to the principles expounded in such a presentation.

3. The proceedings of the various official segments of the Society are published in the JOURNAL as presented by the particular unit for purposes of record.

4. In keeping with its purpose of providing a medium of communication for the members of the Kansas Medical Society, the JOURNAL may publish statements of opinion by individual members or component groups of the Society. Areas available for such statements include the regular Editorial Comment, which may originate from any officer or member of the Society. As encouragement for individual comment on medical matters, material contained in the JOURNAL or Society actions, signed letters are accepted for publication (with the writer's permission). Other commentaries of greater length but based on personal opinion may be acceptable, and these remarks will be identified as to source. Their inclusion in this area of the JOURNAL will indicate that they represent the opinions of the writer and are not necessarily the opinion of the Board, its individual members, or the Society or its officers. The Board considers it an acknowledged expression of its function to edit or reject such material on the basis of liability to legal action arising from it, grossly or destructively derogatory references to any person or legitimate organization, or evident abuse of the privilege of using the JOURNAL for the dissemination of such comment.

5. Advertising revenue is essential to the financial viability of the JOURNAL and the Board shall accept and encourage ethical advertising, both display and classified, relating to legitimate medical items and services or other items and services of value to the medical profession.

6. Items of legitimate interest relating to personal activities and accomplishments of individual physicians are considered proper for inclusion in the JOURNAL. Activities of the Society or its component groups but not relating to official business, items of historical interest such as obituaries, reviews of books of interest to the profession, or other pertinent information constitute valid material for presentation.

7. Informational releases originating from recognized medical groups, such as public health authorities or health education organizations, may be used as space and propriety allow.

8. The JOURNAL shall provide space and assistance wherever possible to auxiliary groups such as the Woman's Auxiliary to the Kansas Medical Society, the Kansas Medical Assistants Society, the Kansas Nurses Association, etc.

Policies

Since the JOURNAL is the organ of the Kansas Medical Society and exists at its pleasure as set forth in the By-Laws, official statements, actions, and policies of the Kansas Medical Society can be expected to govern the management of the JOURNAL, and the JOURNAL cannot ethically adopt an official attitude contrary to any established by the Society.

However, the Board assumes as inherent in its editorial function the privilege and duty to exercise its collective judgment as to policy and decisions affecting this function and the production of the JOURNAL. The benign autonomy implied in this belief is

(Continued on page 437)

Resolutions

To Be Introduced at the Fall House of Delegates, November 18, 1973

RESOLUTION NO. F73-1

(Submitted by the Executive Committee)

Medicredit Bill

WHEREAS, There are Americans who either because of their economic status or due to the nature of their illness find the cost of health care to bring financial distress; and

WHEREAS, It appears that the federal government will institute some form of national health insurance; therefore be it

Resolved, That the Kansas Medical Society strongly urge that national health insurance provide a greater benefit for persons of lesser economic stature; and be it further

Resolved, That the AMA sponsored Medicredit Bill does this more equitably than any other legislation currently before Congress; and be it further

Resolved, That the Kansas Medical Society endorse and urge the passage of the Medicredit Bill; and be it further

Resolved, That copies of this resolution be sent to the Kansas Senators and Representatives.

RESOLUTION NO. F73-2

(Submitted by the Executive Committee)

One PSRO For Kansas

WHEREAS, PSROs are required by law; and

WHEREAS, The Kansas Foundation for Medical Care was established for the purpose of providing peer review such as required under a PSRO; and

WHEREAS, There are many valid arguments favoring a single PSRO for this state; therefore be it

Resolved, That the Kansas Medical Society strongly urges HEW to appoint the Kansas Foundation for Medical Care as the independent contractor to perform professional standards review as required for implementation of PL 92-603 in Kansas.

RESOLUTION NO. F73-3

(Submitted by Jack R. Cooper, M.D.)

Kansas Neurosurgical Society

WHEREAS, The neurosurgeons of Kansas wish to be recognized as a component organized specialty society of the Kansas Medical Society; therefore be it

Resolved, That the By-Laws be amended to add Section 4.58, 17, the "Kansas Neurosurgical Society."

RESOLUTION NO. F73-4

(Submitted by the Southeast Kansas Medical Society, Henry K. Baker, M.D., Delegate)

PSRO and HMO

WHEREAS, The PSRO and HMO laws are in violation of the U. S. Constitution; and

WHEREAS, Legal action is now being brought to test this matter; therefore be it

Resolved, That the Kansas Medical Society, including doctor members and delegates, waste no time at the November meeting planning or discussing these laws.

RESOLUTION NO. F73-5

(Submitted by Section on Ophthalmology, Quentin C. Huerter, M.D., Chairman)

Silver Nitrate

WHEREAS, Since the morbidity from gonorrheal ophthalmia neonatorum is substantial; and

WHEREAS, The incidence of gonorrhea in this state is on the increase; and

WHEREAS, The Credé's method of installation of silver nitrate drops in the newborn's eyes has been effective in preventing this disease; and

WHEREAS, The Section on Ophthalmology of the Kansas Medical Society voted unanimously to continue this method; therefore be it

Resolved, That no action be taken to change the legal status of this silver nitrate treatment, and that the Kansas Medical Society vigorously oppose any legislative attempt to change this status.

RESOLUTION NO. F73-6

(Submitted by the Executive Committee)

Roster

WHEREAS, Resolution 73-20 was adopted by the House of Delegates on Wednesday, May 9, 1973, as follows:

Resolved, That the Roster be amended to include board certification, specialty training and specialty organization other than board certification attesting to clinical competence in accordance with guidelines adopted by the Kansas Medical Society, and be it further

Resolved, That the information on each physician be compiled by the component society in accordance with these guidelines; and

WHEREAS, Technical difficulties discovered in the executive office appear that it is impossible to produce a roster with meaningful information as suggested by the resolution; and

WHEREAS, One sizable component society said they would not supply us with this information; and

WHEREAS, Another sizable component society sent us four typewritten pages on one physician; and

WHEREAS, This would be a sizable cost because the type is retained at the printer and only those lines needing correction are altered. This would require an entire new typesetting; therefore be it

Resolved, That the House of Delegates declare Resolution 73-20 be rescinded, and that a committee be appointed to explore a practical way to provide the additional information that was intended by Resolution 73-20, and that the roster now be sent to the printer to appear as it did last year.

RESOLUTION NO. F73-7

(Submitted by the Executive Committee)

PSRO

WHEREAS, Resolution 49 was adopted by the AMA House of Delegates in New York in the summer of 1973; therefore be it

Resolved, That the Kansas Medical Society endorse and support this resolution:

WHEREAS, Public Law 92-603 (HR 1) as enacted by Congress in 1972 gives over the control of medical practice relating to the delivery of care under certain government welfare medical schemes; and

WHEREAS, This law was supposedly enacted by government to control medical care costs and inflation, when, in fact, the government itself is now spending 406 billion dollars a year or 44 percent of the income of the American people, and has a record of $8,700,000,000 deficit in the past three years; and

WHEREAS, The federal costs (budget) of the Department of Health, Education, and Welfare is now *exceeding* each year the total annual expenditure of the American people for all health care costs; and

WHEREAS, The implementation of Public Law 92-603 or any other law which fails to recognize that excessive spending and bureaucratic interference with a free economy are the real causes of inflation, will fail to control medical care costs and compound the problem; and

WHEREAS, This law will create a new Federal bureaucracy and therefore further add an even larger tax burden for the American people; and

WHEREAS, This law gives direct control to the Secretary of HEW, since that Department pays the bills (Section 1155), writes the rules and compels compliance "in accordance with the regulations of the Secretary . . ." (a phrase used 15 times in HR 1); and

WHEREAS, A National Professional Standards Review Organization which shall be . . . appointed by the Secretary (Section 1163 (a) (1) will have the authority to prescribe and enforce norms and profiles of care, will *supervene over local control* and will decide fines, penalties and sanctions levied against physicians; and

WHEREAS, PL 92-603 will destroy confidentiality because it allows for inspection of not only hospital but also the office records of physicians, which must be made available to the bureaucracy, *including the records of private patients* (current HEW regulations); and

WHEREAS, Physician harassment will occur, since employees and members of PSRO bodies are to be exempted from civil liability for providing information to PSROs; and

WHEREAS, Second-class medical care must inevitably occur as the government will force its concept of *standardization* through the application of computerized norms, national averages and profiles for doctors and patients alike; and

WHEREAS, It further provides in Section 1155 (6) that PSROs "shall" propagandize physicians and the public toward the acceptance of this kind of review control; and

WHEREAS, Under this law regulatory, lay control will be exercised over admissions to hospitals, length of stay, doctors' charges and other services, through the use of PSRO units organized by and acting as federal agents; and

WHEREAS, The medical profession must not and cannot with any rationality bear the responsibility for either the cause of or control of sky-rocketing health care costs, and yet will ultimately receive the blame when the PSRO plan fails to control costs; therefore be it

Resolved, That although it is recognized that repeal or modification of PSRO legislation ultimately may be required to preserve the high quality of patient care, the American Medical Association should oppose any facets of this current legislation which act to the deterioration of quality care, publicize such deleterious facets, and place highest priority on developing and pursuing appropriate amendments to preserve the high quality of patient care.

RESOLUTION NO. F73-8

(Submitted by the Executive Committee)

Non-Physician Individuals Rendering Medical Care

Resolved, That non-physician individuals rendering medical care, including nurse-clinicians, be permitted to act in this capacity only under the supervision and direct sponsorship of an individually named physician who is licensed to practice medicine in the state of Kansas; and be it further

Resolved, That the Healing Arts Act be adhered to, and that the responsible physician bill for the paramedical services rendered.

RESOLUTION NO. F73-9

(Submitted by the Commission for Education)

Postgraduate Education

WHEREAS, The following resolution was filed for information on May 9, 1973; therefore be it

Resolved, That the following resolution be adopted:

(RESOLUTION NO. 73-14)

WHEREAS, It is general knowledge that continued medical education is a necessity to practice good medicine regardless of the specialty; and

WHEREAS, The Commission for Education of the Kansas Medical Society has studied these needs and has recommended that certain postgraduate requirements be met as a requisite to membership in the Kansas Medical Society; therefore be it

Resolved. That the postgraduate study requirements as adopted by the Commission for Education of the Kansas Medical Society be adopted by the Kansas Medical Society as a requirement for continued membership in the Kansas Medical Society.

A. Requirements—150 hours of postgraduate medical education shall be completed by each member every three years as a requisite in continuing membership in the Kansas Medical Society.

1. Accredited education

a. A minimum of 50 hours must be in accredited postgraduate education, such as specialty scientific meetings, medical school sponsored programs, courses approved by AAFP, etc.

b. There will be no limit on credit given for this category, *i.e.,* all 150 hours could be from accredited courses

c. Courses must be approved by the permanent committee appointed by the Commission for Education

d. Credit shall be given on an hour for hour basis.

2. Non-accredited education

a. 100 hours may be non-accredited education such as: (1) Journal reading, (2) Audio digests, (3) Hospital staff meetings, (4) Informal teaching, (5) Publication of papers in recognized professional journals (10 hours credit each), (6) Scientific meetings of state medical society, AMA, and specialty groups.

b. Credit for this category shall be approved by the permanent supervising committee.

B. Administration

1. This program will be under the over-all supervision of the permanent committee

2. Appropriate clerical assistance will be provided in the KMS office

3. The program should be arranged so that one third of the members would meet the requirement each year

4. The requirement for new members shall begin on January 1 of the year following admission to the KMS

5. Requirements shall be on a calendar year basis

6. The individual members shall be responsible for reporting their postgraduate education

7. Appropriate forms shall be furnished by the KMS on a yearly basis

8. Members not meeting requirements shall be notified six months prior to the end of their three-year period

9. Appeals may be made to the KMS Commission for Education.

C. Exemptions

1. Honorary members of county medical societies

2. Members retired from active practice

3. Members in approved residency training programs

4. Special exemptions may be made for long periods of illness

5. Other special exemptions may be granted by the special committee upon application by individual members.

RESOLUTION NO. F73-10

(Submitted by the Commission for Education)

Paramedical Personnel

WHEREAS, The increased use of paramedical personnel by physicians requires the Society to take a stand on this subject; therefore be it

Resolved, That it is the position of the Kansas Medical Society that paramedical personnel do not need to be licensed or registered because they are working under the direction and supervision of licensed physicians; and be it further

Resolved, That billing for services rendered by paramedical personnel is the responsibility of the licensed physician and not the responsibility of persons who do not have a license to perform independent professional services; and be it further

Resolved, That ethics regarding the use of paramedical personnel be resolved by the area peer review committees under the Kansas Foundation for Medical Care.

Personalities—IN KANSAS MEDICINE

A. N. LeMoine, Jr., Kansas City, was the main speaker at a recent meeting of the LaCrosse Lions Club. The topic of the presentation was, "Fight for Sight."

The featured speaker for a program on Arthritis in Clay Center was **Dennis Richards**, Clay Center.

Ned B. Gorrell, Parsons, was the speaker at the annual meeting of the Labette County unit of the American Cancer Society.

Resigning after 40 years of duty as the Ellis County health officer is **G. A. Surface**, Ellis.

Norman G. Marvin, Syracuse, addressed the Hays community fellowship.

S. L. DeBriere, Parsons, has been appointed assistant clinical professor of pediatrics at KUMC.

Participating as speakers for the Medical Assistants annual circuit courses held at Liberal, Wichita, and Topeka were **Carl K. Zacharias**, Dodge City; **James A. Loeffler**, Wichita; **Edward F. Steichen**, Lenora; **Thomas F. Taylor**, Salina; **Eugene E. Kaufman**, Wichita; **Donald D. Hobbs**, Topeka; **James H. Ransom**, Topeka.

William E. Larsen, Kansas City, was the speaker at the annual county-wide meeting of the Atchison unit of the American Cancer Society.

Ernest W. Crow, Wichita, will serve as interim chairman of the department of medicine at the WSU Branch of KUMC, while the search for a permanent chairman continues.

Joyce R. Sumner, Hutchinson, was elected Secretary of the American Society of Anesthesiologists, a 12,200 member organization.

Installed as Fellows of the American College of Surgeons, the world's largest organization of surgeons, were **James P. Newby**, Wichita; **Lawrence W. Hamtil**, Prairie Village; **John D. Douthit**, Manhattan; **William H. Browning**, Wichita; **Russell E. Cramm**, Hays; **Ramon W. Schmidt**, Garden City; **Francisco A. Reyes, Jr.**, Ottawa.

A paper on "Forced Release" was presented at a recent meeting of the Western Conference of Training Schools by **Bruce M. Burdick**, Manhattan.

R. M. Fenton, Garden City, attended a ten-day Physician's Clinic in Omaha.

After practicing for the past 25 years in the Kansas community, **A. F. Schmidt**, Iola, has joined the staff of the V.A. Hospital in Hot Springs, South Dakota.

R. M. Gouldner, Wichita, addressed a naturalization ceremony recently. A naturalized citizen himself, Dr. Gouldner has been a physician in Wichita for more than 50 years.

Leslie E. Becker, Kansas City, was elected president of the Emergency Physicians, Inc. at the group's annual meeting.

Serving as instructors in a course on "Immediate Care of the Sick and Injured" in Edwards County, were **Rene E. Schnoebelen**, **W. Lynn McKim** and **M. Dale Atwood**, all of Kinsley.

Woman's Auxiliary

Katie's Key: International Health Activities

The many key committees in the Medical Auxiliary are bound together with the chain of membership and a concern to do all we can to assist our parent organization. We seek to bring attention to medicine's determination to improve the quality of life of every Kansan by providing even better health education and health care. We focused on AMA-ERF last month. International Health Activities is another key committee on that chain.

Caring, quite properly, involves thinking of others beyond our borders. "Doctors' wives understand how blessed we are," says Dee Cauble, chairman of IHA. Dee is Mrs. Wilbur Cauble who, together with Jo (Mrs. Paul) Lovett, both of Wichita, mastermind the Kansas committee's projects. Both believe that, as doctors' wives, we are bound to stretch hands out to the suffering and the needy, wherever they are. Now in its 13th year nationally, IHA solicits the aid of county Auxiliary groups in worthwhile projects. These include collecting sample drugs from physicians, making quilts, rugs, pillows, mats, dolls, slippers, exercise balls, johnny coats for mission hospital patients, girls' dresses, boys' suits and shirts, bandages, layettes, baby blankets and gowns, as well as collecting old eye glasses, x-ray and EKG equipment, and instruments totaling 349 barrels last year from Kansas alone. It involves collecting and selling old aluminum, cancelled stamps, Betty Crocker coupons, participating in mission projects such as Walk for Mankind, and generally working with other groups to make all of this possible. Overseas recipients are Project Concern, Project Hope, World Medical Relief, and others.

"Fun, satisfaction, and good fellowship result from working together," Dee says. "We are grateful for all the help and interest generated in Kansas. It is truly a state effort!"

National programs include aid to education for children of mission doctors who otherwise would not be able to serve; hospitality for foreign doctors' families visiting in America; overseas volunteers; Pen Pals; projection of international health-thinking through information and programs; and sharing our scientific books. Individual families in Kansas have been part of these programs too. Yes, we have a key to concern for those in need throughout the world . . . International Health Activities!

Katie Keys

115th Annual Session

KANSAS MEDICAL SOCIETY

May 5-8, 1974, Topeka

MODERN ASPECTS OF TRAUMA

1. **Aeromedical Transportation of the Injured**
2. **Medico-Legal Aspects of Trauma**
3. **Symposium on Fat Embolism**

The 1974 meeting will be held in conjunction with the following specialty societies:

KANSAS SOCIETY OF PATHOLOGISTS
KANSAS OBSTETRICAL AND GYNECOLOGICAL SOCIETY
KANSAS ALLERGY SOCIETY
KANSAS SECTION ON EAR, NOSE AND THROAT
KANSAS SOCIETY OF ANESTHESIOLOGISTS

The D. C. Line

Editor's Note: The Editorial Board feels that the Kansas Medical Society would profit by maintaining contact with Representative Bill Roy, both as a source and interpreter of political activities on the national medical front. Accordingly, we are initiating a monthly column written by Dr. Roy. It is hoped that this will not only provide the service intended, but invite a response of readership either through the JOURNAL or to Dr. Roy directly.

Last month, Senators Long and Ribicoff introduced a new health insurance bill, S.2513, the Catastrophic Health Insurance and Medical Assistance Reform Act.

The proposal has three parts. The first—Catastrophic Health Insurance Program (CHIP)—provides for payment out of the *Social Security trust fund,* for medical expenses exceeding $2,000 in one year, and for hospitalization over 60 days. The second part provides for payment out of the general funds of all health care for the poor. The third part makes it possible for private health insurance companies to submit policies for federal certification.

It is with regret that I conclude that the Long-Ribicoff health insurance bill is not in the best interest of the people or health professionals.

Why? For the simple reason that it is the first step to paying for the health care services for all people of all ages by the federal government. The federal government already pays for health care services for the elderly (Medicare) and shares in the payment for health services for the poor (Medicaid). It does not now pay for health care services for people of all ages who can afford to pay for care themselves.

If we, the people of this nation, look to the federal government to pay for all health care services, we will find that there will not be enough dollars to pay for health care—to the very great detriment of us all. We, as physicians, know that we cannot provide quality health care without adequate dollars.

The immediate answer to my above stated objection is that Long-Ribicoff will pay federal dollars only for catastrophic illness (now defined as medical expenses greater than $2,000 in one year). This is correct. But Long-Ribicoff establishes a payment principle—payment for health care for people of all ages out of the Social Security trust fund. Once this financing-payment mechanism is established, it will be with us for decades, perhaps centuries.

If this proposal becomes law, all that is left to be done is the changing of the benefit package. The $2,000 will be lowered to $1,500, then to $1,200, etc. CHIP will prove to be a prophetic acronym.

Are there facts to support this foreboding and forewarning? Yes—the following.

1. Unemployment Insurance, Workman's Compensation, Social Security, and Medicare are all examples of the fact that once established, financing-payment mechanisms for these programs remain the same—benefits may and do change (always increasing) but the financing-payment mechanisms have been—and will likely be—with us forever.

2. When everyone pays in—as occurs in Long-Ribicoff—everyone wants to benefit to some degree. All working Americans will pay an increased payroll tax to provide the proposed additional health insurance. Only a few (perhaps one in twenty) will benefit annually. More will wish to benefit. To accomplish this, benefit packages will be increased; the minimum figure will be lowered. This is the same reason that catastrophic health insurance has not been widely sold by private health insurance companies.

3. There are substantial numbers of health planners in this country who sincerely believe that all health care services should be paid for by the federal government. It is their stated intention to take what federal payment they can get now for health care, and then to increase benefits and broaden eligibility to eventually cover all health care costs.

Catastrophic health insurance is needed. Everyone should have it. Individuals and families should not face poverty and the welfare rolls because of the cost of an illness which literally bankrupts them. The issue is how should catastrophic health insurance be financed.

The answer of Senators Long, Ribicoff, and their other co-sponsors is that financing and payment should be by the federal government, through the Social Security trust funds.

If you, my fellow physician, believe that it is in the best interests of your patients to have their health care paid for by the federal government, then you should support the Long-Ribicoff proposal.

If you, my fellow physician, believe as I do that reliance on federal tax funds will very quickly mean inadequate dollars for health care, to the great detriment of the American people, then you should oppose the Long-Ribicoff proposal.—*W.R.R.*

House of Delegates

November 18, 1973

See Page 417

"THE KILLERS"

The first of five 90-minute television programs under the inclusive name, "The Killers," will be presented over the facilities of Kansas' PBS stations: KTWU-Channel 11, Topeka, and KPTS-Channel 8, Wichita, on Monday, November 19. The subject of the first presentation is "Heart Disease" and it will emphasize the fact that heart ailments in various forms afflict all ages. The individual stations will feature local programs following the nationwide showing in order to focus on the facilities as well as the problems of heart disease in the community.

The second program, "Inborn Genetic Defects," will be shown over the same stations on Monday, December 17. National advisors in the preparation of this segment include Dr. Leon Rosenberg, Yale University School of Medicine; Joseph Mori, National Foundation-March of Dimes; Dr. Park Gerald, Harvard Medical School; Dr. Alexander Bearn, Cornell University Medical College Department of Medicine; and Dr. Robert F. Murray, Jr., Howard University College of Medicine.

The genetics presentation will discuss a variety of genetically-induced disorders as well as some diseases in which genetic factors may play a role. Directions of genetic research will be examined in addition to the presentation of established information in the field. Ethical and moral features of both current knowledge and prospective advances will also be examined. As with the first program, community-oriented programs will be offered by the local stations following the main telecast.

December 17—Inborn Genetic Defects
January 14—Pulmonary Disease
February 11—Trauma
March 11—Cancer

26th Annual

MIDWEST CANCER CONFERENCE

CENTURY II MARCH 29 & 30, 1974 WICHITA, KANSAS

"CURRENT CONCEPTS IN THE CLINICAL MANAGEMENT OF CANCER"

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Columbia University
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M. D. Anderson Hospital
University of Texas Medical Center
Houston, Texas

LEO P. CAWLEY, M.D.
Department of Pathology
Wesley Medical Center
Wichita, Kansas

PROGRAM

9:00 A.M. "PROSPECTS FOR THE 'CURE' OF DISSEMINATED HODGKINS DISEASE"—Charles Coltman, Jr., M.D.
10:15 A.M. "CURRENT CONCEPT IN THE MANAGEMENT OF PROSTATIC CARCINOMA"—John B. Wear, Jr., M.D.
11:30 A.M. Luncheon—Guest Speaker: Leo P. Cawley, M.D.—"LABORATORY TESTS FOR THE EARLY DIAGNOSIS OF CANCER: A STATUS REPORT"

FRIDAY AFTERNOON SESSION
This session will start promptly at 1:00 P.M. and adjourn at 5:00 P.M. "CURRENT CONCEPTS IN THE TREATMENT OF BREAST CANCER, A SYMPOSIUM"—Cushman D. Haagensen, M.D., Vera M. Peters, M.D., George Crile, Jr., M.D., Anthony Curreri, M.D.

FRIDAY EVENING
6:00 P.M. to 7:30 P.M. "VISIT WITH THE PROFESSOR"—This is a special session to be held in the Crown Room of the Regal Inn.

SATURDAY MORNING SESSIONS
9:00-9:50 A.M. **Section A**—for internists—"CURRENT CONCEPTS IN THE THERAPY OF LEUKEMIA AND LYMPHOMA"—Vera M. Peters, M.D., Charles Coltman, Jr., M.D., Jay P. Sanford, M.D. **Sections B & C**—for surgeons and gynecologists—"URINARY DIVERSION BECAUSE OF MALIGNANT DISEASE: WHEN, HOW, AND WHY"—John B. Wear, Jr., M.D.
10:15 A.M. **Section A**—continuation—"CURRENT CONCEPTS IN THE THERAPY OF LEUKEMIA AND LYMPHOMA"—Drs. Peters, Coltman, Sanford. **Section B**—"CURRENT CONCEPTS IN THE THERAPY OF FEMALE GENITAL TRACT CANCER"—Gilbert Fletcher, M.D., James L. Breen, M.D. **Section C**—"CURRENT CONCEPTS IN THE SURGICAL THERAPY OF CANCER"—George Crile, Jr., M.D., Anthony Curreri, M.D.

The Professional Education Committee of the Kansas Division of the American Cancer Society invites all physicians in the entire midwest to attend a two-day graduate course on cancer. Free registration.

MEDICAID RECIPIENTS TO BE SPOT-CHECKED

HEW's Social and Rehabilitation Service has proposed a regulation requiring states to inform Medicaid providers and recipients of the criminal, anti-fraud provisions of a new law affecting the medical assistance program.

Medical assistance, generally referred to as Medicaid, is a grant-in-aid program in which the federal and state (and sometimes local) governments share in the cost of medical care for persons with low income. It is authorized by the Social Security Act.

The proposed regulation—published in the *Federal Register*—is entitled "Fraud in the Medical Assistance Program."

Public Law 92-603 (Social Security Amendments of 1972) imposes penalties of $10,000 or imprisonment for up to one year, or both, for anyone soliciting, offering, or accepting kickbacks, bribes, or rebates; for anyone concealing or withholding information affecting a person's right to any benefit or payment with the intent to defraud; or for anyone converting benefits or payments to improper use.

Also, misrepresentation of health, safety, and operating conditions in health care facilities to permit them to qualify for Medicaid payments would be subject to a penalty of not more than six months' imprisonment, a fine of up to $2,000, or both.

Fraudulent acts in the Medicaid program had been covered previously only by state laws and general federal laws on fraud.

The proposed regulation would note additionally that health care providers and facilities that are ineligible to participate in Social Security's Medicare program because of fraudulent activities could not receive federal Medicaid funds.

Further, the regulation would expand a previously issued regulation on fraud. States had been required to spot check Medicaid recipients to see if medical services for which bills were submitted had been performed. Under the proposed regulation, states with federally financed, computerized management systems would be required to notify all Medicaid patients of the specific services for which providers were submitting bills, the names of such providers, the dates of the services, and the amount of the payments to the providers. Thus, Medicaid recipients would help states make sure that Medicaid funds were spent for services given.

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Symposia:

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MANHATTAN—St. Mary Hospital
Dec. 4—Metabolism
Jan. 8—Therapy
Feb. 5—Cancer
Mar. 5—Cardiovascular
Apr. 2—Gastroenterology
Apr. 30—Trauma

WINFIELD—William Newton Memorial Hospital
Dec. 6—Metabolism
Jan. 10—Therapy
Feb. 7—Cancer
Mar. 7—Cardiovascular
Apr. 4—Gastroenterology
May 2—Trauma

HUTCHINSON—St. Elizabeth's Mercy Hospital
Dec. 5—Metabolism
Jan. 9—Therapy
Feb. 6—Cancer
Mar. 6—Cardiovascular
Apr. 3—Gastroenterology
May 1—Trauma

EMPORIA—Newman Memorial County Hospital
Dec. 7—Metabolism
Jan. 11—Therapy
Feb. 8—Cancer
Mar. 8—Cardiovascular
Apr. 5—Gastroenterology
May 3—Trauma

WESTERN CIRCUIT

CONCORDIA—Cloud County Community Jr. College
Dec. 4—Gastroenterology
Jan. 8—Cardiovascular
Feb. 5—Trauma
Mar. 5—Cancer
Apr. 2—Metabolism
Apr. 30—Therapy

DODGE CITY—Dodge City Community College
Dec. 6—Gastroenterology
Jan. 10—Cardiovascular
Feb. 7—Trauma
Mar. 7—Cancer
Apr. 4—Metabolism
May 2—Therapy

COLBY—Ramada Inn, I-70 at Colby Exit
Dec. 5—Gastroenterology
Jan. 9—Cardiovascular
Feb. 6—Trauma
Mar. 6—Cancer
Apr. 3—Metabolism
May 1—Therapy

HAYS—Hadley Memorial Medical Center
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Jan. 11—Cardiovascular
Feb. 8—Trauma
Mar. 8—Cancer
Apr. 5—Metabolism
May 3—Therapy

Registration Fee: Six sessions, $60.00. (Single session, $20.00.)
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For program announcement and information, write: DEPARTMENT OF POSTGRADUATE MEDICAL EDUCATION, University of Kansas Medical Center, Kansas City, Kansas 66103.

PSRO INTERNAL PROBLEMS

William I. Bauer, M.D., has resigned as director of the controversy-ridden Professional Standards Review Organization (PSRO) program, expressing dissatisfaction with the PSRO organization setup.

The surprise step-down was a shock to the top officials at HEW who have been reeling from the loss of other high officials upset over the lengthy reorganization of the health activities at the HEW department.

Charles Edwards, M.D., Assistant HEW Secretary for Health, interrupted a planned business retreat to hurry back to Washington when news of the resignation filtered out. He called a news conference but then cancelled it after the reporters had shown up. Dr. Edwards was in conference with HEW undersecretary Frank Carlucci at that time.

The PSRO program is a particularly sensitive one to be subject to the inevitable repercussions and criticisms that follow a resignation. Members of the Senate Finance Committee have been taking a hard line on involvement of state medical societies in the PSRO review of institutional care under Medicare and Medicaid. Some physicians groups and state societies, and the PSRO advisory committee, have urged a broader authority for state societies. In general, HEW and Dr. Bauer had appeared to be attempting a middle course.

Furthermore, the gearing-up for the intricate and complicated program has been a mammoth task for Dr. Bauer. The 48-year-old Dr. Bauer was named to the PSRO post last March after a career as a practicing internist in Greeley, Colo.

Other HEW officials who have resigned in the past several months are Gordon McLeod, M.D., director of the Health Maintenance Organization (HMO) program, and Arthur Lesser, M.D., head of Maternal and Child Health Services.

In a statement, Dr. Bauer said the administration has made a "significant commitment to PSRO but that commitment has not been translated into action. . . ."

"This extremely complex program with ramifications at all levels of medical care has been provided with limited resources and those resources that were made available could not be effectively administered and utilized because of the organizational structure," Dr. Bauer said.

According to an HEW spokesman, the resignation stemmed from a dispute between Drs. Bauer and Edwards over organizational control of the PSRO program. Dr. Bauer was said to believe that he could not exert meaningful authority under the present setup in which much of the field work for PSRO, involving hundreds of physicians, would not come under his line control but under the Bureau of Quality Assurance. Dr. Edwards, the spokesman said, contended that Dr. Bauer would still have the say-so, but Dr. Bauer obviously disagreed.

Underlying the dispute, apparently, has been the effort of Dr. Edwards to pry PSRO control away from Social Security and Social and Rehabilitation Services, present overseers of Medicare and Medicaid, and to give the Health Department clear jurisdiction in PSRO.

Under the reorganization, 50 physicians at Social Security and 150 in the Health Services Administration are assigned to PSRO but not directly under Dr. Bauer who had 36 staff positions.

There was no indication from Dr. Bauer of any philosophical differences with the administration over how PSRO would function at the local and state level.

HMO LEGISLATION

The House has approved legislation that will provide federal funds to start a limited number of experimental Health Maintenance Organizations (HMOs) over a five-year period to the tune of $240 million. The Senate version of HMOs, passed months ago, would provide $805 million over the same period. House and Senate conferees must now resolve the differences.

The compromise bill voted by the House calls for spending $60 million this fiscal year, the Administration figure. The bill meets many objections raised to the original measure by the Administration and the American Medical Association.

Though no specific number limitations was set in the House bill, the limit of authorizations to $240 million will provide an effective ceiling on the number of HMOs which could be established. The House Commerce Committee estimated the legislation would be used to bring to the operating stage approximately 100 new HMOs.

The bill has a flat five-year cut-off for the HMO program.

Unlike the Senate bill, the House legislation does not pre-empt state laws that restrict formation of HMOs. The reason given by the House Commerce Committee was "the rapid change already underway in state legislation designed to remove these barriers." Approximately 20 states have adopted legislation specifically authorizing HMOs.

The bill limits grants or contracts for planning and initial development costs by prohibiting this assistance after 1976. Initial development assistance would be prohibited after 1977.

Loans and loan guarantees for initial operation costs are authorized except that loan guarantees could be provided only if the HMO will serve residents of a medically underserved area. The bill has no authority for loan guarantees for construction projects.

For grants and contracts for feasibility studies, initial planning and initial development costs, the bill would authorize $40 million for fiscal year 1974, $45 million for fiscal year 1975, and $50 million for fiscal year 1976. In addition, it would authorize $55 million for fiscal year 1977, for grants and contracts for initial development costs. The bill would authorize $20 million for fiscal year 1974, and $30 million for fiscal year 1975, to be appropriated to the loan fund.

The bill, unlike the original subcommittee bill, has no authority for demonstration grants and contracts for enrollment of the indigent, for providing service in rural medically underserved areas, and for enrollment of high risk individuals. There also is no authority for special project grants and contracts, for grants for HMO management training, and for program evaluation.

Provisions for protection against insolvency of HMOs, against the cost of providing unusual amounts of health services or of providing out of area health services, and protection against unusual losses were not contained in the final bill. Also deleted were provisions which authorized technical assistance and consultative services to aid in the planning or development of an HMO.

For the Record

(Continued from page 427)

deemed necessary for effective execution of its duties.

The Board believes it is part of its editorial function to give full consideration to the publishing of constructive opinions of individual members although such opinion may be contrary to established policy. Such opinions will be identified as to source and should not be construed as representing official Board opinion. The privilege to express such opinions should be available to any member of the Society, including officers or members of the Editorial Board, providing the distinction between personal opinion and official Society policy is maintained.

In the event the Council is of the opinion that the Board or any member is derelict in his duty or, in official capacity as a Board or indivdiual member, acts contrary to official Society policy, the power of the Council to remove such individuals is constitutionally recorded.—*D.E.G.*

A NEW FEDERAL AGENCY

HEW Secretary Caspar W. Weinberger announced the establishment of a new health agency to deal with the sociomedical problems of alcohol abuse, drug abuse, and mental health and illness.

Called the **Alcohol, Drug Abuse, and Mental Health Administration,** it becomes the sixth agency of the Public Health Service (after the Food and Drug Administration, the National Institutes of Health, the Health Services Administration, the Health Resources Administration, and the Center for Disease Control) and its third-largest with an approximate fiscal 1973 budget of $650 million. ADAMHA will consist of three institutes: the existing National Institute on Alcohol Abuse and Alcoholism, the National Institute of Mental Health, and the new National Institute of Drug Abuse.

Secretary Weinberger said that a search for an individual with the necessary medical, scientific, and administrative skills to head the Alcohol, Drug Abuse and Mental Health Administration has begun. In addition, Secretary Weinberger announced that ADAMHA's National Institute of Drug Abuse will be headed by Dr. Robert DuPont, director of the White House Special Action Office on Drug Abuse Prevention. Dr. Bertram Brown and Dr. Morris Chafetz will continue in their present capacities as director of NIMH and director of NIAAA respectively, the Secretary said.

The new agency was recommended to Secretary Weinberger by Assistant Secretary for Health Dr. Charles C. Edwards, following the report of a special Task Force. Dr. Edwards pointed out that the new organization gives appropriate visibility and leadership to the high priority programs of drug and alcohol abuse, as well as mental health and illness. These three areas of concern, he said, "span both the health and human service systems. While they clearly relate to health concerns, they also interact closely with the courts, schools, and social service agencies."

The Journal accepts short classified advertising from the members of the Kansas Medical Society without a charge. These ads run in three consecutive issues of the Journal and are keyed with a correspondence number. All replies are forwarded immediately to the advertiser. Other brief classified ads are accepted from members of the medical profession only upon approval of the Editor or Editorial Board.

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AN EFFORT TO REDUCE MALPRACTICE LOSSES

A special effort to diagnose and treat the problem of escalating medical malpractice losses has been announced by one of the leading sources of medical liability insurance, St. Paul Fire and Marine Insurance Company, through initiating a program designed to help physicians prevent incidents which lead to malpractice claims and to speed up the disposition of cases.

The major elements of the program are:

1. Arbitration will be advocated as a speedier and less costly claims settlement practice. Doctor-patient and hospital-patient arbitration agreements will be sought in jurisdictions showing interest and where arbitration is a practical approach. Model agreements have been prepared by The St. Paul. The company believes arbitration decisions should be based on medical expertise.

2. Special materials alerting physicians to current trends and unusual exposures will be produced. These will be aimed at helping physicians prevent injuries and at assisting in physician-to-patient communications.

3. Claims data will be computer programmed to aid in the identification of medical liability trends and recurring causes of losses. Information obtained will be used in developing loss prevention information for physicians.

4. Formation and use of medical review panels by state medical associations will be more strongly encouraged and assisted where requested. The company endorses review panels of medical personnel designed to aid the company in determining whether a departure from the standard of medical care occurred.

5. Techniques and materials to enable physicians to obtain good informed consents from their patients will be given special attention. The company believes that a well informed patient is less likely to assume that less than successful results from surgery or other treatment automatically means the physician was negligent.

As one of the leading insurers of the medical profession, The St. Paul's claims experience indicates there are a number of things physicians can do in daily practice and patient communications to reduce the potential for malpractice claims.

For malpractice claims where there is disagreement over whether negligence actually occurred or the amount of damages, arbitration procedures could reduce the time otherwise spent waiting for litigation to move through the usual legal process.

Announcements

Professional meetings, conferences, and postgraduate courses of national importance are listed for the DOCTOR'S CALENDAR. *Notice of the session is posted in advance to allow the physician time to make preparations.*

NOVEMBER

Nov. 12-17 Laryngology and Bronchoesophagology, at the Eye and Ear Infirmary, 1855 W. Taylor, Chicago 60612.

Nov. 14-17 Automotive Medicine 17th Annual Conference, Hilton Inn West, Oklahoma City. Write: Page Waller, Oklahoma State Department of Health, NE 10th & Stonewall, Oklahoma City 73105.

Nov. 15 Therapy in the Rheumatic Diseases; 4th Annual Arthritis Symposium, St. Francis Hospital Nurses Auditorium, Wichita. Write: Frederick Wolfe, M.D., Chairman, Professional Education Committee, Kansas Chapter Arthritis Foundation, 345 N. Hillside, Wichita 67214.

Nov. 18 Kansas Medical Society House of Delegates, University Ramada Inn, Manhattan.

Nov. 26-29 Care of Injured (American College of Surgeons), University of Texas SW Medical School, Hilton Inn, Dallas.

Nov. 29-Dec.1 Virology and Immunology in Human Cancer, Waldorf-Astoria Hotel, New York. Write: S. L. Arje, M.D., American Cancer Society, 219 East 42nd St., New York 10017.

DECEMBER

Dec. 1-5 American Medical Association, Anaheim, Calif.

Dec. 1-6 American Academy of Dermatology, 32nd Annual. Palmer House, Chicago. Write: F. A. J. Kingery, M.D., 2250 N.W. Flanders, Portland 97210.

Dec. 7-8 Ophthalmology and Otolaryngology, Kansas City Society annual meeting. Write: 3036 Gillham Road, Kansas City, Missouri 64108.

Dec. 8 American College of Sports Medicine, Central States Annual. KU Campus, Lawrence. Write: Harley Hartung, M.D., Central Missouri State University, Warrensburg, Missouri 64093.

Dec. 12-14 Care of Injured (American College of Surgeons), Brown Palace, Denver. Write: Dr. John A. Boswick, 4200 East 9th, Denver 80200.

JANUARY

Jan. 19-23 American Academy of Allergy, Americana Hotel, Bal Harbour, Fla. Write: J. O. Kelley, 225 E. Michigan, Milwaukee 53202.

Jan. 19-24 American Academy of Orthopaedic Surgeons, Fairmont Hotel, Dallas. Write: C. V. Heck, M.D., 430 N. Michigan, Chicago 60611.

Jan. 22-24 Pediatric Pulmonary Course, 3rd Annual, sponsored by the Louisiana Thoracic Society and the American Lung Association, New Orleans. Write: W. F. Raymond, 333 St. Charles, Suite 1504, New Orleans 70130.

Jan. 25-27 AMA National Leadership Conference, Mariott Motor Hotel, Chicago. Write: AMA Public Affairs Division, 535 N. Dearborn, Chicago 60610.

FEBRUARY

Feb. 11-14 American College of Cardiology, Hilton and Americana, New York. Write: W. D. Nelligan, 9650 Rockville Pike, Bethesda 20014.

Feb. 13-16 National Association of Medical Examiners, Statler Hilton, Dallas. Write: W. G. Eckert, M.D., St. Francis Hospital, Wichita 67214.

University of Kansas:

Nov. 5-8	*Internal Medicine*
Nov. 28-30	*Medical Technology*
Jan. 16-24	*Gynecology and Obstetrics*, Rio de Janeiro, Brazil
Jan. 31	*Diagnostic Cytology*

For further information, write the Department of Postgraduate Medical Education, KUMC, Kansas City, Kansas 66103.

University of Colorado:

Jan. 21-26	*General Practice Review*
Feb. 4-6	*Thromboembolism*
Feb. 11-15	*Newborn Radiology Seminar*

For further information, write the Office of Postgraduate Medical Education, University of Colorado School of Medicine, 4200 E. Ninth Ave., Denver 80220.

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1973

VOL. LXXIV
NO. XII

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Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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Volume LXXIV, Number 12—December, 1973

The JOURNAL of the KANSAS MEDICAL SOCIETY

Contents for December

Scientific Articles

Human Mycoplasma Infection: Neurological Complications—Subramoney Nagaswami, M.D., Stuart W. Twemlow, M.D. and John Kepes, M.D., Topeka 441
Minor Surgery Center: An Answer to Escalating Costs—M. Robert Knapp, M.D., Pere A. Owen, M.D., LaRue W. Owen, M.D., Robert Wallace, M.D. and Courtney Clark, M.D., Wichita 446
Permanent Transvenous Pacemakers: Non-Electrical Complications and Hazards—Willy Pereira, M.D. and James C. Mershon, M.D., Wichita 450
Operant Conditioning Procedures: Conditioning in Schizophrenic Work—Donald R. Reimer, M.D. and Thomas J. Fitzgerald, Ph.D., Topeka 456

Student Thesis

Sydenham's Chorea: Current Concepts and Literature Review—Robert E. Fast, M.D., Kansas City, Kansas 459

Socio-Economics

Supplemental Security Income Program—Dwight Lawson, M.D., Topeka 463

Miscellaneous

Advertisers 19
Announcements 12
Book Reviews 6
Classified Advertisements 19
D. C. Line 471
Editorial Comment: Dilemma *du jour* 466
Index to Volume LXXIV 477
Information for Authors 455
KUMC Postgraduate Education 449
Leadership Conference 445
Midwest Cancer Conference 17
Month in Washington 14
New Members 455
Obituaries 468
Official Proceedings, 1973 Fall House of Delegates 472
Personalities 470
President's Message 465
Woman's Auxiliary 469

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Scientific ARTICLES

Human Mycoplasma Infection

Neurological Complications

SUBRAMONEY NAGASWAMI, M.D., STUART W. TWEMLOW, M.D. *and* **JOHN KEPES, M.D.,** *Topeka*

MYCOPLASMA associated with human disease was first reported by Dienes & Edsall,[1] who isolated mycoplasma from a suppurating Bartholin's gland abscess in a female laboratory worker in 1937. One year later, Reimann[2] introduced the term "atypical pneumonia," having found meningism associated with pneumonia in 5 of 103 patients. It was not until 1950, however, that Eaton[3] first suggested that atypical pneumonia was not necessarily caused by a virus. In 1962, Chanock[4] succeeded in cultivating the organism and demonstrated that it was mycoplasma; M. pneumoniae. Since this early research, M. pneumoniae remains the only mycoplasma, a fully proven pathogen of man, although recently other antigenically distinct types have suggestive epidemiological evidence that they are associated with exudative pharyngitis. Mycoplasmas have been recovered from patients with arthritis, arthralgia,[5] and chronic bronchitis.[6] Skin lesions recorded include maculopapular rashes, erythema nodosum, and Stevens-Johnson syndrome.[7] Coombs positive hemolytic anemia is the most common hematologic disorder encountered, but thrombocytopenia and coagulation defects have also been reported.[8] Raynaud's phenomenon, pericarditis, myocarditis, myringitis, perception deafness, and nonbacterial otitis media[8] have all been attributed to M. pneumoniae infection. Mycoplasmas have been recovered from leukemic bone marrow[8] and have been associated with abnormal human diploid cell development in cases of Down's syndrome.[9] Association of mycoplasma with antibodies to human brain[10] and to other tissue antigens[11] might suggest finer etiological studies focused on the role of mycoplasma in the broad category of autoimmune disease.

A case of distinct association with M. pneumoniae and related to the complication of transverse myelitis.

The literature stresses that M. pneumoniae may be symptomless in the human respiratory tract, and that bronchopneumonia develops in only 3-10 per cent of infected persons.[12] Often, a transient, minor flu-like illness may develop with most infections being non-epidemic; however, Evatt *et al.*[13] recorded a severe epidemic involving 55 college students, which suggested that intimate contact was required for infection, contrasting markedly with easy spread from influenza and adenovirus sources. Feizi[8] reported an epidemic in a general practice in 1964-1965, with much more prolonged convalescence required.

Yesnick,[14] in 1956, gave the earliest comprehensive review of an association between primary atypical pneumonia (PAP) and complications involving the

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central nervous system (CNS). While the incidence of neurological complications of PAP is given by Yesnick as 1 per cent, this figure may be low, since the true nature of the infection may be masked by the more dramatic nature of the complications and, thus, when reviewed, is easily missed. In the 19 case reports, approximately one-third of the patients died in a spectrum of complications including meningitis, meningoencephalitis, Guillain-Barré syndrome, hemiplegia, cranial nerve palsy, and transverse myelitis. Of the four cases of transverse myelitis, none presents adequate information for more than a mildly suggestive association. Incidentally, Yesnick also notes that most reports were from epidemics seen in various military installations during World War II when close contact would favor rapid spread of infection.

Other reports reflect an increasing interest in M. pneumoniae as an etiological agent in Guillain-Barré syndrome,[16] and a suggestion of its involvement in acute cerebellar ataxia.[17] M. pneumoniae has also been reported in association with bacteria in patients with septic meningitis.[18]

Case Report

The patient was a 25-year-old white, married male employed as a chemical laboratory technician by a local rubber company. He was well until the last of March 1971, when he developed cough, malaise, and a mild sore throat of two or three days' duration; he recovered and was asymptomatic for approximately one week. Over a 24-hour period prior to admission on April 6, the patient noticed some numbness, tingling, and faint weakness with equilibratory instability in both legs. He attempted to go to work on the morning of admission, but was referred immediately for treatment.

On investigation, the patient gave no pertinent past or family history other than recent exposure to rubeola four weeks previously. He had no history of vaccinations or of the infectious exanthemas of childhood and was in excellent health all his life, including two years of military service. Examination revealed that the patient was unable to stand or walk; his temperature was 104 F (40 C); pulse rate, 102; blood pressure of 150/90 mm Hg; with rapid irregular respirations. Ear, nose, and throat were normal on admission except for doubtful icterus of sclerae bilaterally persisting for ten days. The skin showed no eruptions, and the lungs were clear to auscultation. There were no palpable lymph nodes. Findings in the CNS included gross transverse myelitis with loss of all modalities of sensation below the D-11 segment of the spinal cord. The knee and ankle reflexes were hyperactive, and there were bilateral Babinski's signs at that time. He also had moderate nuchal rigidity, and straight leg raising was limited to 40°. Cerebrospinal fluid (CSF) examination performed immediately showed a pressure of 130 mm H_2O; no manometric block; white blood cell count (WBC) 64 cu mm; with a differential of 92 per cent lymphocytes, 8 per cent polymorphonuclear, and a total protein content of 107 mg/100 ml. The fluid showed no growth on culture for yeast and bacteria. In addition, he was found to have an atonic neurogenic bladder. A catheter was inserted on admission.

Laboratory studies disclosed the following values: hemoglobin, 14 gm/100 ml; mean corpuscular hemoglobin concentration (MCHC), 43 per cent; WBC, 4,600/cu mm, with a differential of 61 per cent neutrophils, 38 per cent lymphocytes, and 1 per cent monocytes. Sedimentation rate was 24 mm/hour. Peripheral blood serology was normal, as were urinalysis and serum electrolyte levels. Chest x-ray was reported as normal, although serial comparison suggests a mild tracheobronchitis.

Hospital Course

During the next 24 hours, the myelopathy ascended into the midcervical segment of the spinal cord giving a sensory level of C4-C5, and was accompanied by a serious impairment of the patient's ability to breathe. A tracheostomy was carried out on April 7, with the institution of respirator assistance. His temperature continued to be elevated, ranging from 101 to 103 F, but the trend to normal appeared on April 29. One day after admission, the patient erupted a generalized cutaneous rash most marked over the anterior thorax and abdomen, which continued for four days. During this period, further secondary complications included paralytic ileus, requiring levine tube aspirations, and pneumonic consolidation of left lower lobe with partial pneumothorax. He was critically ill until May 1, continuously requiring intravenous feeding, positive pressure respiratory assistance, gastric aspiration, and indwelling urinary catheter. Ten days after admission, the neurological signs receded from the midcervical region to the region between D7 and D6, and remained at this level thereafter with a total absence of all sensory modalities below that level. The initial spastic paralysis of the lower limbs became flaccid with a degree of muscle wasting and total absence of all lower limb reflexes. The abdominal reflexes were absent from the third day after admission. Motor power in the upper limbs improved with some residual interosseous atrophy. Cranial nerves were normal throughout. Signs of meningism disappeared after the tenth hospital day, and reexamination of CSF on May 13 showed pressure of 120 mm H_2O; no manometric block; total protein of 290 mg/100 ml; lymphocytes, 8/cu mm; colloidal gold curve, 00000000; syphilis test was negative. Rubeola complement-fixing antibody tests on CSF and serum were less than 1:4. Frequent blood counts during this period reflected the secondary bacterial pneumonia with elevation of sedimentation

rate up to 49 mm/hour, with changes in serum biochemistry reflecting the critical nature of the illness. The level of serum glutamic oxaloacetic transaminase (SGOT) was 145 mg/100 ml; bilirubin totalled 1.6 mg/100 ml, 0.9 mg/100 ml direct and 0.7 mg/100 ml indirect; total serum protein, 6.1 gm/100 ml; globulin, 3.2 gm/100 ml. Serum electrolyte levels were remarkably stable. Investigations and history were considered to exclude the traditional causes of transverse myelitis including postvaccinal, trauma, spinal vascular disease, systemic lupus, carcinoma, glandular fever, and narcotic ingestion. A diagnosis of virus infection of the nervous system was made.

The patient was transferred to the Veterans Administration Hospital on May 31, 1971, at which time his sensory level had improved to D8-D9. He had an indwelling catheter necessitated by an atonic neurogenic bladder. The external rectal sphincter was atonic, and he was incontinent of feces.

Therapy

The patient had received gamma globulin injections for the first days after admission, and subsequently variable broad spectrum antibiotics for the respiratory complication.

Following transfer, the patient continued to improve to a sensory level of D-10, and he continued an active physical therapy program. Complement fixing antibody test results became available indicating M. pneumoniae, 1:64 (May 7), later increasing to 1:128 (June 9), and 1:256 (June 30). Insignificant levels were recorded for rubeola, herpes simplex, Western equine encephalomyelitis, St. Louis encephalitis, mumps, lymphocytic choriomeningitis, polio types 1, 2, 3, and psittacosis. No virus was isolated from the spinal fluid or serum. On the weekend of September 10, when home on pass, the catheter became obstructed, and the patient returned to hospital with a temperature of 103 F. Despite immediate parenteral antibiotic therapy, his temperature rapidly rose to a confirmed level of 108 F, after which he became comatose and developed gram-negative septicemic shock which failed to respond to intraveneous Keflex and steroids. He died on September 14.

Pathology

Pathohistological examination of CNS identified as the cause of death septicemic shock with a gross degree of cerebral edema. The upper cervical and high dorsal spinal cord region showed no definite abnormalities; however, a section of high dorsal and middorsal region showed such an extreme degree of softening that the dura mater and pia mater were not removed in order not to destroy all the remaining spinal cord architecture. From approximately D4-5 level of the dorsal cord down to the lumbar region, advanced degree of necrotic softening of the entire spinal cord could be seen *(Figure 1)*.

Figure 1. Low-power views of spinal cord illustrating increasing autolysis with necrotic softening maximum in the lumbar region.

Histological examination demonstrated a patchy admixture of gitter cell reaction with areas of persistent necrosis without inflammatory or phagocytic activities. Microscopic cavitation was seen in several areas, and there was no sign of vascular causation *(Figure 2)*.

The presence of coagulation type necrosis with anuclear dead nerve cells still present without any phagocytic or inflammatory activity around them appears to be a subtype of necrotizing myelitis first reported by Lowenberg, DeJong and Foster, 1941.[19] In the Foix-Alajouanine type of necrotizing myelitis, the venous occlusive component is recognizable and prominent with a more gradual onset of spinal cord problems. The type of nonvascular necrotizing myelitis seen in this case appears to be a final common path for a number of etiological agents (in this case as mycoplasma pneumoniae infection) where the morphology of CNS lesion appears to be quite independent of the nature of the preceding disease *(Figure 3)*.

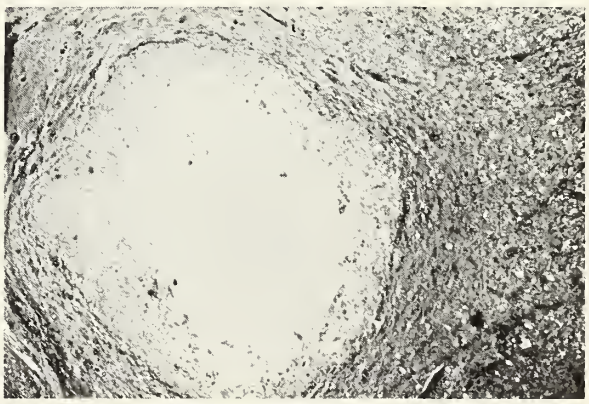

Figure 2. Microscopic view of dorsal spinal cord in transverse section.

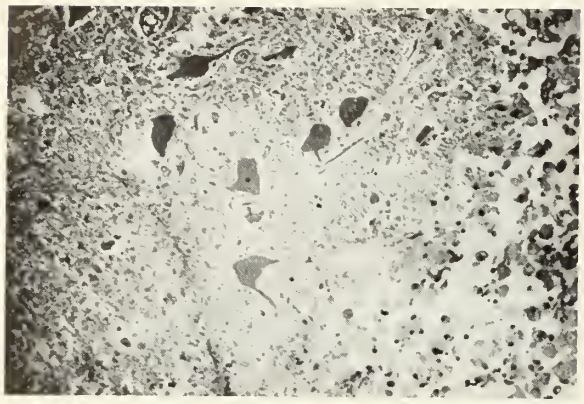

Figure 3. Microscopic view of the dorsal spinal cord showing coagulation necrosis with anuclear dead nerve cells without phagocytic or inflammatory activity.

Discussion

The clinical course of the patient suggested a viral illness without radiological evidence of primary atypical pneumonia, a point heavily emphasized in the literature.[12] The high fever, normal total WBC with a relative lymphocytosis, the premorbid flu-like illness with an elapsed period from onset of the respiratory problem to the appearance of the transverse myelitis of ten days support the available literature[14]; however, incubation periods of up to 23 days have been reported for polyradiculitis. While an increase in complement-fixing antibody is sometimes seen at the time of onset of the illness,[12] in this case the rising titre was not dramatic until the second month of illness. The complement-fixing method is useful and reliable, but less sensitive than other methods.[21] CSF findings in the literature show little consistency aside from the increase in protein and the absence of manometric block. The suspicion of nonbacterial etiology was heightened by lymphocytic preponderance in CSF cells in the absence of tuberculosis, although the literature reports polymorphonuclear dominance in some cases.[14] The CSF protein continued to rise after the cellular reaction settled, although the reported rate of rise in other neurological complications of M. pneumoniae infection varies. Signs of meningeal irritation which are commonly reported in association with more specific clinical entities in M. pneumoniae infection, and were noted transiently in this case. The clinical and laboratory findings of an aseptic CSF indicated an acute and devastating illness. The autopsy findings showed a severe necrotizing myelitis. These phenomena are strikingly out of proportion to the intensity of the initial respiratory complaint, and are possibly compatible with an allergic, autoimmune process. An alternative suggestion of a direct neurotoxic effect has some analogy in the experimental literature. Neurotoxin production in M. neurolyticum, the cause of an acute encephalopathy in rats, has been studied.

While the evidence in this case strongly supports a direct etiological relationship between M. pneumoniae and the resulting transverse myelitis, the statistical significance of pleuropneumonia-like organisms (PPLO) in etiology of transverse myelopathies is far from clear. In a comprehensive review of the literature since the 1800s, it has been suggested that about one third of all cases may well have a nonbacterial etiology. It was noted that even the very early authors suggested "allergic" causes.

Summary

The first proven case of necrotizing myelitis after Mycoplasma pneumoniae in a 25-year-old white male was reported. He had an acute upper respiratory infection with an exanthematous eruption. This was followed in 10 days' time with a transverse myelitis, resulting in complete section of the cord at D-11 level. In 24 hours, it ascended to C-4 level and stabilized later at the D-6 level. He developed complications of respiratory distress needing tracheostomy, neurogenic bladder, paralytic ileus, and lung atelectasis. He died four months later of gram-negative septicemia.

Extensive laboratory work on blood, urine, and CSF was done with negative results, except for leucopenia. Spinal fluid showed normal pressure, with no block, increased lymphocytes and increased protein which subsequently increased further. Blood, CSF culture for bacteria, fungus, serial complement fixation tests for viruses of all types were negative except for mycoplasma, which rose from a titre of 1:16 to 1:256 in four weeks. Autopsy findings to substantiate the diagnosis were given. A review of the neurological complications of mycoplasma and a discussion of the causation of necrotizing myelitis were given.

Acknowledgments

Mrs. N. Vaughn, Medical Librarian, V.A. Hospital, and Mrs. L. Marvin, Secretary, Neurology Service, V.A. Hospital, for literature search work and assistance.

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(Continued on page 462)

LEADERSHIP CONFERENCE

February 1-3, 1974

The Crown Center Hotel—Kansas City, Missouri

All members, and especially the presidents and secretaries of component county medical societies, are earnestly urged to attend this first annual conference which will include seminars and sections on administrative and organizational problems at the local level.

The speakers will include:

Malcolm C. Todd, M.D., Long Beach, Calif.—President-Elect, American Medical Association.

Jerome P. Lysaught, Ph.D., Rochester, New York—Director, National Commission for the Study of Nursing and Nursing Education.

Bill Roy, M.D., Washington, D. C.—U. S. Congressman, Kansas Second District.

Bernard P. Harrison, J.D., Chicago, Ill.—Director, Medical Practice Division, American Medical Association.

William O. Rieke, M.D., Kansas City, Kansas—Vice-Chancellor for Health Affairs, University of Kansas Medical Center.

Robert Haakinson, Ph.D., Philadelphia, Pa.—Associate Dean, School of Business Administration, Temple University.

The program will center around organizational and administrative problems encountered in local societies in communication with KMS and utilization of available information; legislative update—federal and state; the changing role of nursing in today's medicine; AMA and its relationship to the local practicing physician; and other topics.

More information will be forthcoming by mail and in subsequent issues of the JOURNAL. If you have questions, please contact the KMS Executive Office, either by calling collect, (913) 235-2383; or by writing, 1300 Topeka Avenue, Topeka, Kansas 66612.

ALL KMS MEMBERS ARE INVITED TO ATTEND

Minor Surgery Center

An Answer to Escalating Costs

M. ROBERT KNAPP, M.D., PERE A. OWEN, M.D., LARUE W. OWEN, M.D., ROBERT WALLACE, M.D. *and* **COURTNEY CLARK, M.D.,** *Wichita*

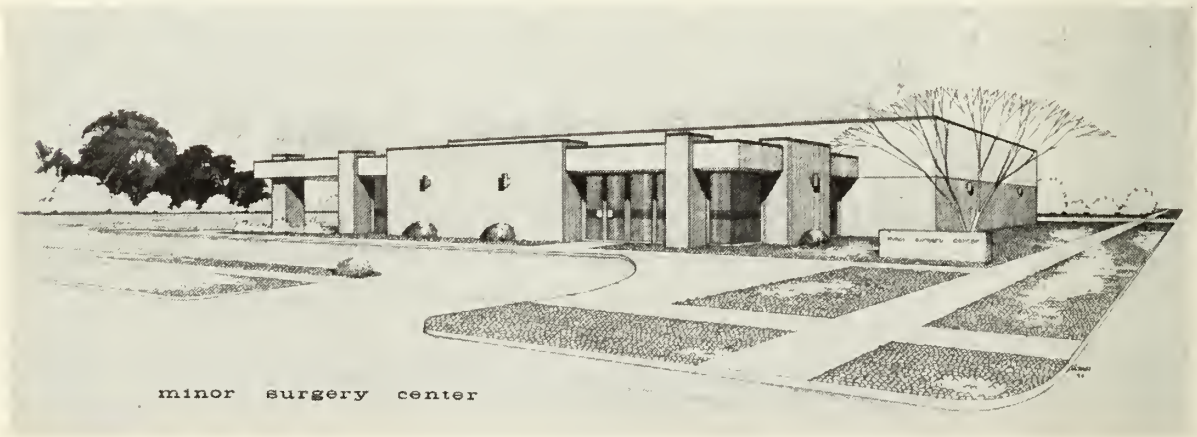
A preliminary report of a soon-to-be opened Minor Surgery Center in Wichita. Designed to bridge the gap between the physician's office and the hospital, it is expected to relieve some of the hospital load, save the patient money, and save the physician time.

TWO PHOENIX anesthesiologists, Drs. John Ford and Wallace Reed, opened a short-stay surgical facility on February 12, 1970. They named it Surgicenter, and located it across the street from Good Samaritan Hospital in Phoenix, Arizona. The Surgicenter is owned and operated by these two men.

Surgicenter has catalyzed the thinking of many people in the health care field in the United States. Until the time it opened, the bulk of the experience in outpatient surgery had been gained at George Washington University in Washington, D. C., and UCLA in Los Angeles, California. Now that Surgicenter has been functioning for more than three years, it can be shown that it has met its objectives with resounding success. To quote *Medical Economics* of May 28, 1973, "Some 5,500 outpatients treated in the latest full year at Surgicenter in Phoenix, Arizona, saved an average of more than $130 apiece. Comparisons with costs for the same procedures on an inpatient basis were made by insurers and a hospital unconnected with Surgicenter."

Inspired by the example being set in Phoenix, Minor Surgery Center of Wichita, Inc. became a reality. The medical profession of America has for many years been challenged to "do something" about the high costs of medical care. This appeared to be an appropriate response to that challenge, the rendering of high quality surgical care at lower cost to the patient, an appropriate private practice solution to one of the most nagging of medicine's problems. Dr. Russell Lee was quoted in *Medical World News* in its issue of July 11, 1969: "The modern hospital is rapidly becoming an intensive care unit with all the fantastically expensive equipment and procedures that are required. It costs too much: It costs too much because many who are hospitalized do not need all this and should be cared for in much less expensive surroundings. Thus, the hospital must be surrounded with a number of closely associated special purpose facilities." On January 6, 1971, H. Charles Abbott, president of Blue Cross of Southern California, told Senate investigators in Washington, D. C. that "the acute general hospital is sometimes used when the patient's level of care can be provided adequately in less expensive and less intensive care facilities."

The vast increase in the demands placed upon the modern hospital is rapidly making the cost of minor surgery prohibitive. This, in terms of dollar expense,



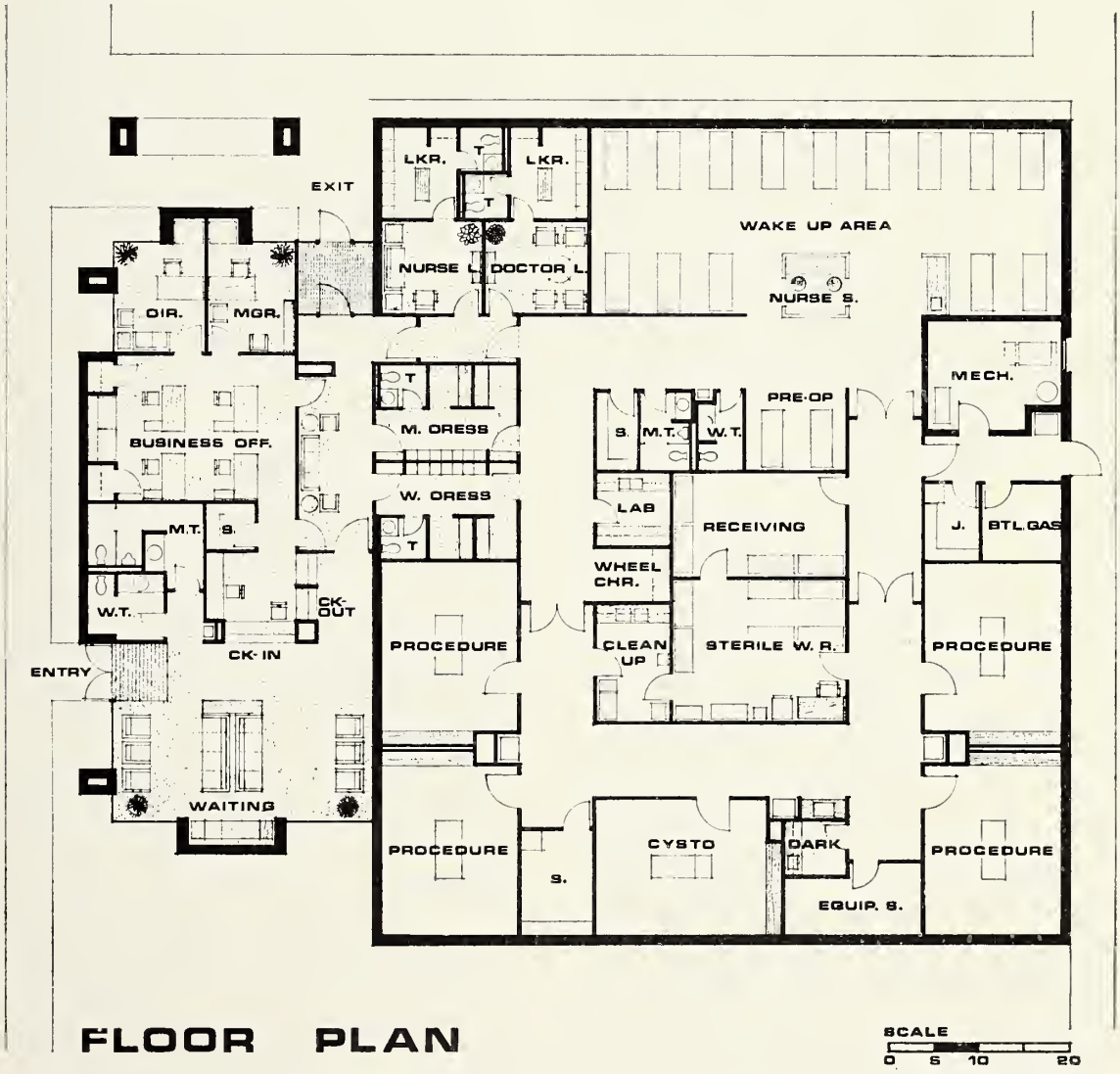
inefficient use of professional time, and patient inconvenience. Much of this three-fold cost is directly attributable to the fact that many patients are hospitalized in elaborate facilities when they do not need the highly sophisticated service of a modern hospital. Their unnecessary presence in the hospital, in turn, imposes a serious burden on the patients who do need the full resources of our modern hospitals.

The dollar cost of medical care requires no documentation. Hospital admissions for simple surgical operations inevitably result in unnecessary charges for rooms and other services inherent in such admissions. This problem has become a substantial element in the health care costs experienced by a community.

The additional costs in terms of the consumption of inefficiently utilized professional care and patient time are less obvious than the hospital cost but are no less severe. These costs relate primarily to the matter of congestion in the surgical suites.

Serious surgical cases and protracted procedures should and do command priority over the kinds of cases that will be performed at Minor Surgery Center. Under present hospital conditions, the minor surgical admittee often experiences delays in his admission to hospital, last minute revision of surgical schedules, and postponement of operations. These are all serious in terms of the patient's morale and the surgeon's time.

Bed space is limited and additional bed space is very costly to the community. If a suitable outpatient surgical facility were available, beds occupied now by patients who do not need in-hospital care would free beds for patients who do need hospitalization. Such competition for bed space and for money to provide more beds is not medically or economically sound. When bed space is not available, the cost to the public is measured in terms of delayed treatment for all patients, which means a lower than acceptable quality



FLOOR PLAN

of medical care.

One particular advantage to the Minor Surgery Center of Wichita, Inc. involves the care of children. Acute hospitalization, in a strange environment, including parent abandonment at night, is often a very frightening experience for the child.

The Minor Surgery Center has been planned to minimize this psychological impact. The child will be brought to the Center approximately one hour before surgery, taken to a holding area specifically designed for the small patient, later receive premedication, and be allowed to remain with the parents until the premedication has had its effect. Following surgery and the recovery room stay, the child will again be united with his parents and return to the familiar environment of home. Much has been recently written concerning the participation of parents in the care of the child. No doubt, the parents are as well equipped to comfort and attend the child during the convalescent post-surgical period as the same post-convalescent child placed on a busy post-surgical floor. The dangers of cross contamination and infection should also be minimized.

It was against this background that Minor Surgery Center of Wichita, Inc. evolved. The building that will house the Minor Surgery Center is under construction at 810 North Lorraine, Wichita, near all three major hospitals. The building contains five operating rooms and a recovery room to accommodate 16 patients. All the necessary supportive functions can be found within the slightly more than 9,000 square feet of space. The design of the building was carefully studied by the architects and the authors, who have accumulated many years of experience working in operating room surroundings. It is designed for patient and physician convenience.

Ford and Reed summed up the purposes of such ambulatory surgical facilities . . . "to make the 'ambulatory patient' a matter of greater concern; to streamline the delivery of his medical services; to reduce the cost of his care."[1] The intent of Minor Surgery Center is the same.

The Congress of the United States received a report from the Comptroller General in December 1972, titled "Study of Health Facility Construction Costs." In this report, he stated that the "Appropriateness of medical care depends on using the right facility for the right patient at the right time, . . . appropriate use of ambulatory care facilities, where personal health care services not requiring an overnight stay are rendered, also results in more efficient use of inpatient facilities and thereby reduces the need for these facilities, . . . surgical procedures performed in ambulatory surgical centers also have saved one to two patient days: One hospital projected annual savings of 18,000 acute beds nationally as a result of its experience with the ambulatory surgical facility. *Our analysis tends to confirm this estimate.*"

In late 1970, Minor Surgery Center approached both the Medical Society of Sedgwick County and the Health Planning Council of South Central Kansas with its contemplated aims. We made a formal request for approval of our intent. Both organizations made exhausting inquiry of the ambulatory surgical concept and on December 15, 1970, the Medical Society of Sedgwick County "recommended endorsement of a 'free standing' outpatient surgical facility in the Wichita community." On May 4, 1971, after being approved by both an Ad Hoc Committee and the Sedgwick County Associate Council, the Health Planning Council of South Central Kansas recommended "the implementation in Wichita of a free standing Surgicenter and recommend implementation by the hospitals of their proposals."

In 1971, the Kansas Legislature amended the Hospital Licensing Act with Senate Bill No. 331 to include ambulatory surgical centers. The Act was further amended in the 1973 Kansas Legislature to more precisely define such facilities. Minor Surgery Center is complying with all the provisions of the law and will be licensed when construction is finished.

With the aid of the Health Planning Council of South Central Kansas, an "agreement of conditions for the establishment of an ambulatory surgical center" was negotiated. This was signed on April 2, 1972. The agreement calls for medical audit by a team of physicians appointed by the Medical Society of Sedgwick County. These physicians are to accept their appointment in writing, stating that they have no economic or vested interest in the facility. Their duties are to recommend and enforce standards of care and recommend practices designed to avoid overutilization. The Medical Society of Sedgwick County appointed a seven-man committee; all have agreed to serve.

A Community Advisory Committee will be appointed within 60 days of the commencement of operation of Minor Surgery Center. Such committee has not yet been appointed but will include at least six citizens representing varied areas of the community.

The agreement also mandated transfer agreements between Minor Surgery Center and the local hospitals. This requirement was met by correspondence from the hospitals indicating their willingness to accept patients from Minor Surgery Center.

On August 24, 1972, a contract between Kansas Blue Cross and Minor Surgery Center was signed. Kansas Blue Cross will cover all services rendered to its subscribers in Minor Surgery Center. The charge levied by Minor Surgery Center is one which is all-inclusive for each procedure. In this way Kansas Blue Cross, other insurance carriers, the individual patient, and each surgeon will know *in advance* exactly what the cost of a given procedure will be. This is guaranteed predictability at its best.

Physician's fees are not a part of this all-embracing facility charge.

The Health Insurance Association of America has given verbal assurance that the Center has complied with the requirements of the commercial carriers and that they will recommend coverage of services in this facility to their member companies.

In October 1972, among the amendments of the Social Security Act that were passed was one having to do with Section 222 of the law. It specifically authorized the Secretary of HEW to experiment with reimbursement of ambulatory surgical centers under Title 18. Minor Surgery Center has indicated its interest in participating in such an experiment. HEW is now drawing the necessary guidelines and we have every reason to believe that our facility will be one of those chosen.

We have discussed with our colleagues in much detail the *raison-d'être* of our facility. Their suggestions have been welcomed and will continue to be. Minor Surgery Center is designed to serve the patient and the surgeon in the most harmonious surroundings possible.

It is interesting to note the interest that the nurses of the community have manifested in the concept of ambulatory surgery. They have, with few exceptions, volunteered their endorsement of it. Further, there have been numerous requests from them for employment. They have very kindly volunteered recommendations on equipment and procedural matters, and these have been given every consideration in the evolution of policy and inventory lists.

The Minor Surgery Center will be an asset to the Wichita community. It will bridge the gap existing between the physician's office and the hospital. Procedures too small for the hospital and too large for the office can be conveniently and readily done there. This facility is designed to provide minor surgery care to patients of all ages. In this regard, it is to be thought of as a specialty center where special attention is given to the infant as well as the aged. The patient has assurance that his health insuror will provide him with coverage for this service. The surgeon has the assurance of ready convenience with minimal paper work. Quality control is assured through the Surgical Advisory Committee of the Medical Society of Sedgwick County. The community has the advantage of another health care facility which will be available to it in times of emergencies or disasters.

We have responded to the challenge laid down to the medical profession to reduce costs while maintaining quality care. It is our intention to do so. We also wish to demonstrate the viability of the free standing ambulatory surgical facility.

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Permanent Transvenous Pacemakers

Non-Electrical Complications and Hazards

WILLY PEREIRA, M.D.* *and*
JAMES C. MERSHON, M.D.,† *Wichita*

CARDIAC PACING is a well-known, established, and accepted treatment modality for patients subject to serious or fatal cardiac disturbance of rhythm or conduction, specifically for those suffering from significant consciousness impairment or organ insufficiency related to compromised cardiac output. However, it is important to emphasize the possibility of potential and well-known complications associated with this tool.

The records of 150 patients at St. Francis Hospital, who underwent permanent transvenous pacemaker implantation from November 1970 to February 1973, were reviewed in a retrospective mode with particular interest on those with documented features supporting non-electrical pacing complications: x-rays, electrocardiograms, cultures, operative reports, and other data.

Case One

This is the case of an 85-year-old nun, who had had a permanent implanted cardiac pacemaker using transvenous endocardial electrodes, and was admitted because of hemolytic anemia secondary to chronic staphylococcus aureus coagulase positive infection at the site of the implanted generator unit. She was found on admission to have a hemoglobin count of 7.5 gm/100 ml. She had been on long-term antibiotic treatment with methicillin and oxacillin. This was the second implant at the same site.

Physical examination revealed the following: temperature, 98.8F (37.2C); pulse rate, 68 beats per minute; respirations, 18 per minute; blood pressure, 188/70 mm Hg. The cardiac pacemaker unit was felt over the upper lateral aspect of the right chest anteriorly without evidence of fluid in the pocket. The scar was well healed. The heart sounds were distant without abnormalities.

Laboratory studies revealed the following values: hemoglobin, 7.5 gm/100 ml; hematocrit, 20%; red blood cell count (RBC), 2.02 million/cu mm; white blood cell count (WBC), 5,400/cu mm; total iron binding capacity, 260 μg/100 mg. Hemoglobin electrophoresis showed A1, 95%; A2, 5%. Reticulocyte count was 20.6% (normal range is 0.3 to 1.5%). Osmotic fragility was within normal limits. Red cell survival was reported as being markedly shortened—six days compared to the normal of 28-35 days. External counting carried out over the spleen and liver showed spleen/liver ratios of 0.6:1.0.

This case represents a thrombotic complication involving the pacemaker catheter.

Subsequent course consisted of exteriorization of the cardiac pulse generator and multiple drainage of infected cardiac pacemaker pulse generator pocket. The patient was treated with oxacillin and prednisone. The hemoglobin on discharge was 10.5 gm/100 ml; hematocrit, 31%; reticulocyte count, 5%.

This case of an abscess at the site of the implanted generator unit illustrates infection as a complication.

Case Two

This patient, an 84-year-old man, related having recurrent episodes of syncope. One month prior to this admission, the electrocardiogram showed second degree heart block with episodic atrial fibrillation and bradycardia, and a transvenous permanent pacemaker was implanted. Blood pressure was 212/90 mm Hg on admission, and the pulse rate was 104 beats per minute. Later in the hospital course, the ventricular response of the atrial fibrillation dropped to 56 beats per minute. Examination revealed no pacing of extra cardiac structures. A pacemaker sound, which increased with inspiration, was heard in systole. No murmurs or gallops were present.

A chest x-ray on admission showed the tip of the pacemaker catheter to be in the right atrium, explaining the lack of pacing *(Figure 1)*. A temporary pacemaker was inserted through a cut-down in the left antecubital area and the permanent electrode was repositioned in the right ventricle.

This case represents an example of dislocation of the pacemaker electrode.

Case Three

This 56-year-old white male was readmitted in August 1972, because of pacemaker malfunction. He

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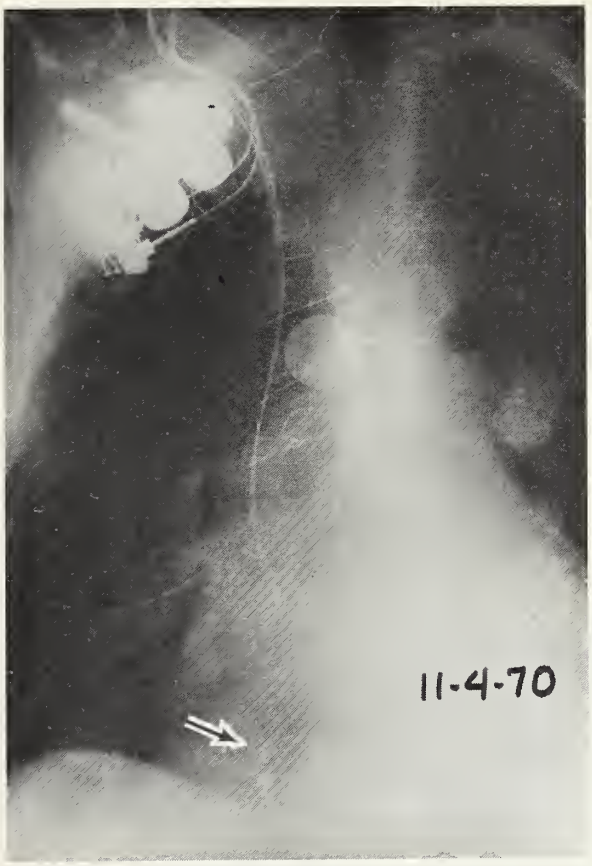

Figure 1. Chest x-ray, Case 2: bipolar electrode coiled in right atrium (arrow).

had a history of permanent implantation of transvenous pacemaker with endocardial bipolar electrode in February 1971, for syncope and third degree A-V block. During this admission, he experienced spells of lightheadedness and dizziness, and his pulse rate had decreased to 34 beats per minute, from 70 beats per minute.

On physical examination, the temperature was 98F; pulse rate, 36 beats per minute; respirations, 18 per minute; blood pressure, 130/70 mm Hg. The electrocardiogram showed a ventricular rate of 40 beats per minute, with the pacemaker blips showing no relation to the segment of electrocardiograph (QRS). The patient was alert, complaining of intermittent periods of dizziness and lightheadedness. The cardiac pacemaker was felt subcutaneously in the upper left pectoral area, and the scar over the same area appeared to be well healed. The heart sounds were reported as being slightly distant, but no abnormal sounds were heard. A chest x-ray showed loss of insulation of the pacing electrode *(Figure 2)* and, subsequently, a second pacing catheter was inserted. The loss of insulation of the pacing catheter graphically shows another mechanical complication of permanent pacing.

Case Four

This is the case of a 46-year-old black male, admitted with the chief complaint of right arm and facial swelling present for one month prior to admission in November 1971. Fourteen months previously, a permanent transvenous pacemaker was implanted because of complete heart block, following the sequential appearance of left axis deviation and right bundle branch block. Physical examination revealed the blood pressure to be 130/90 mm Hg; respirations, 20 per minute; temperature, 98.2F; pulse rate, 72 beats per minute. It was noted that the neck veins showed mild venous engorgement, with lack of increased jugular venous distention on the right side with the Valsalva maneuver. Over the right pectoral area, a subcutaneous cardiac pacemaker unit was palpated. An early grade I/VI systolic ejection murmur was heard at the apex and over the left sternal border. Examination of the extremities revealed the right arm to be swollen with normal pulses over brachial and radial arteries, and mild collateral venous circulation over the upper right arm and shoulder.

A bilateral subclavian venogram was done, which revealed complete occlusion of the right subclavian, right external jugular, and right innominate vein *(Figure 3)*. There were also multiple filling defects on the pacing electrode in the superior vena cava and down to the level with the junction of the right atrium.

Complete blood count showed hemoglobin, 14.1 gm/100 ml; hematocrit, 42.6%; WBC, 6,100/cu mm with 9% eosinophils, 51% neutrophils, 30% lymphocytes, and 10% monocytes. Coagulogram showed a platelet count of 150,000/cu mm, and a normal morphology. IVY bleeding time in the left arm was 5 minutes, with a normal of 1-9 minutes. Lee White clotting time was 5 minutes (normal, 5-15 minutes). Prothrombin time was 10 seconds, with a normal control of 10 seconds. Prothrombin consumption time was 23 (normal, 25 seconds or longer). Activated partial thromboplastin time was 24.5 (control time, 29 seconds); fibrinogen level was 300 mg/100 ml (control level was 258 mg/100 ml, and normal limits are 170 to 410 mg/100 ml). Clot retraction was complete at 30C at 24 hours, and only partial retraction was seen at room temperature at 24 hours. Quantitation of immunoglobulin showed 1800 IgG, 200 IgA, 84 IgM—all were within normal limits. Total protein was 6.5 gm; albumin, 2.93 gm; globulin, 1.39 gm. Treatment consisted of intravenous heparin and subsequent sodium warfarin anticoagulation. Bilateral upper extremity venograms four months later still showed complete obstruction of the right innominate vein and multiple filling defects adherent to the electrode in the superior vena cava, but numerous collateral channels had appeared.

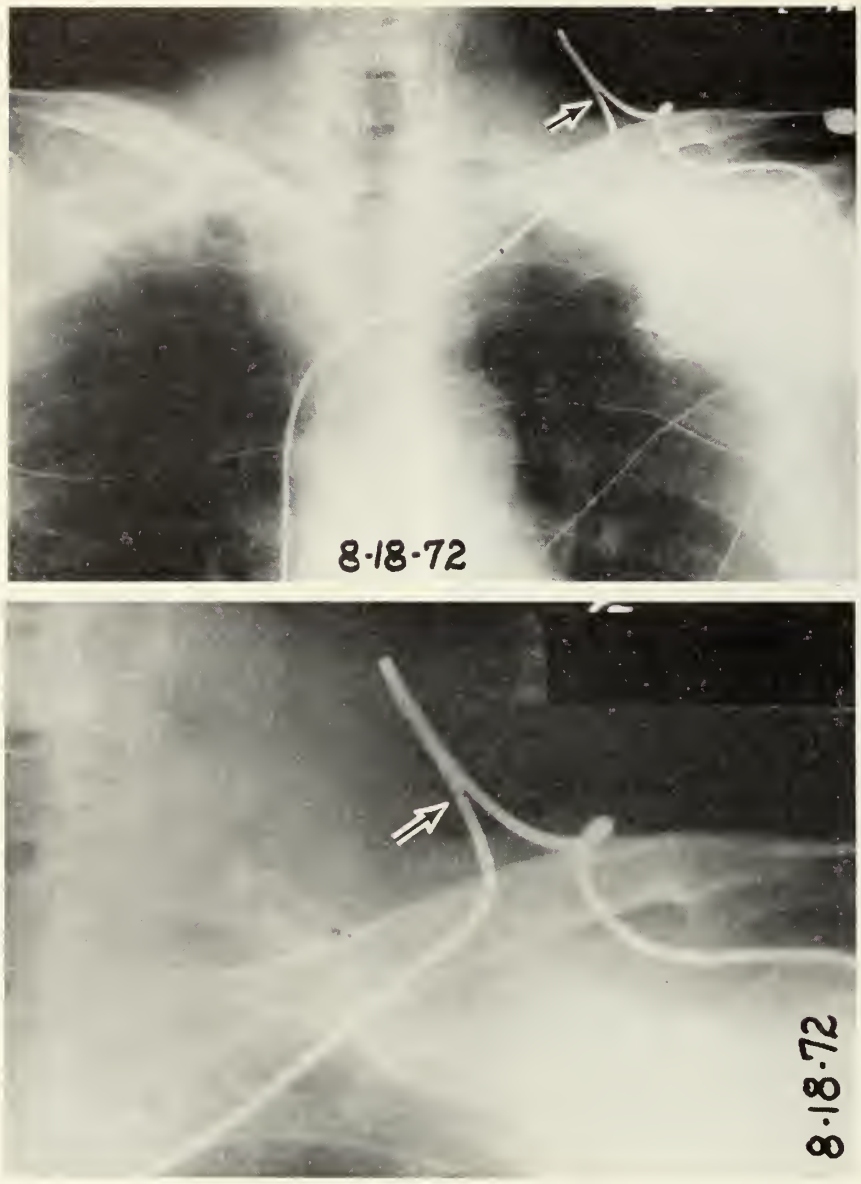

Figure 2. (*a* and *b*) Case 3: Disruption of insulation of pacemaker catheter (arrow).

Results

One hundred and fifty records of patients with permanent transvenous pacemakers were reviewed. Five cases (3%) with non-electrical complications were discovered, as follows: one case of infection of the pulse generator unit; two cases of dislodgement of the pacing catheter; one case of disruption of pacing catheter insulation; one case of vein thrombosis of the pacing catheter.

Discussion

Table I outlines the potential complications associated with permanent transvenous pacing. The object of this paper has been to approach the second category, complications non-electrical in origin related to pacemaker insertion.[1-3] The displacement of the catheter electrode tip is usually an early complication leading to loss of pacing, effectiveness, intracardiac malposition, and placing of structures extraneous to the heart. Penetration and perforation of the heart can occur and instances of pacing of the diaphragm, pericardial irritation, and cardiac tamponade have been reported.[2-6] In an attempt to avoid this complication, a satisfactory anatomic and electrical position for the catheter electrode tip involves the following:

a) Placement into the apex of the right ventricle in an area not in contact with the valvular tissue or

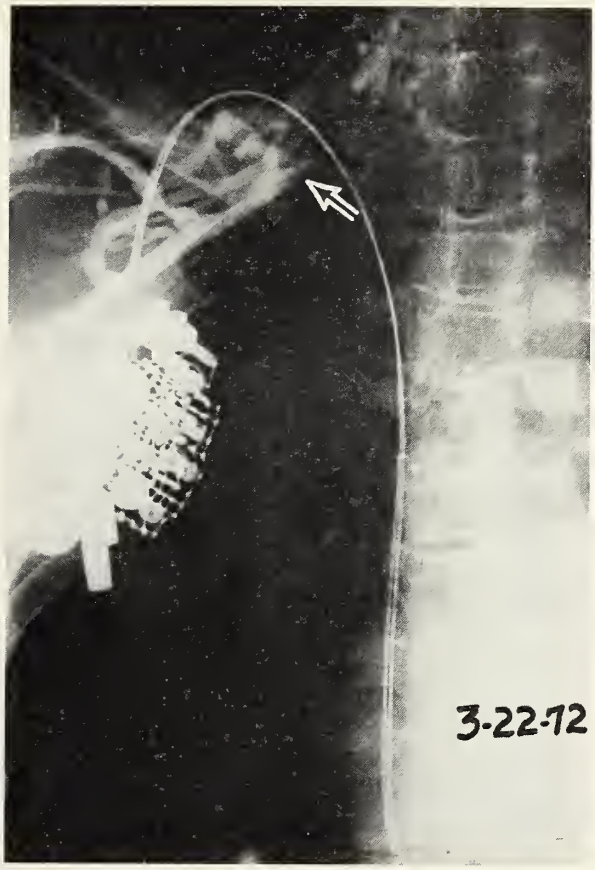

Figure 3(a)

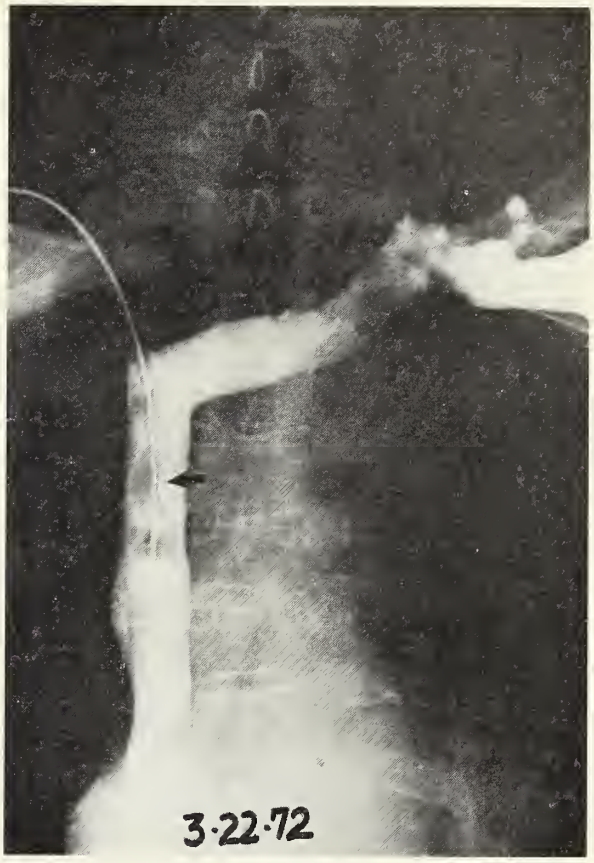

Figure 3(b)

Figure 3. (a) Case 4: Venograms reproduction shows venous obstruction of right subclavian vein. *(b)* Thrombus surrounding catheter in superior vena cava (arrow).

chordae tendineae.[1]

b) Placement beneath trabeculae where it will be held securely until it is surrounded by fibrous tissue and cannot be ejected.[1]

c) Placement in the area of lowest threshold of stimulation consistent with electrode being used.[7]

d) Avoiding increased tension or excessive curves in placing electrode catheter.

Also, bleeding at the site of the pulse generator can cause subcutaneous hematoma with local pain, discoloration, and, if overlooked, in extreme cases, anemia.

Findings which indicate penetration are usually changes in the electrical axis of the pacing, intermittent diaphragmatic pacing, presence of a pericardial friction rub, extra cardiac auscultatory clicks, and cardiac tamponade secondary to perforation of the myocardium. When penetration and perforation by pacemaker endocardial electrodes is suspected, a radiographic demonstration by spot film could be helpful; a separation of less than 3 mm between the electrode tip and the cardiac fat indicates that penetration has occurred.[5]

Laceration of the tricuspid valve caused by removing a pacemaker wire has been reported. This occurs as a result of the underlying fibrous encapsulation of the catheter electrode in the right ventricle and in the tricuspid valve, and this complication should be remembered when one is considering removing a catheter electrode.[8]

Systolic clicks and scratchy systolic murmurs with inspiratory augmentation have been heard in patients with endocardial electrodes. These most often are of no hemodynamic significance.[9]

Sepsis at the site of the pacemaker unit generator implant has been reported.[10] Sometimes it is necessary to perform appropriate drainage or replacement of the unit to another location to avoid more serious complications, such as infective thrombophlebitis of the subclavian vein and bacterial endocarditis.[11] Systemic antibiotics and local irrigations with topical antibiotics should also be employed. Attempts to treat conservatively without replacement have been successful.[10]

Infection at the site of the pulse generator implant, when a unit has been reimplanted at the same site,

TABLE 1

PACEMAKER COMPLICATIONS

1. ELECTRICAL (Related to Improper Pacemaker Function)
   a. Defective-Depletion Pulse Generator
   b. Increase Endocardial Threshold
   c. Dysrhythmia
   d. External Electrical Interference
   e. Pacing of Extra Heart Structures
2. NON-ELECTRICAL
   a. Dermatitis
   b. Bleeding
   c. Air Embolism
   d. Thrombosis
   e. Acoustic Phenomenon
   f. Penetration-Displacement Perforation
   g. Cardiac Tamponade
   h. Infection

is manifested usually by mild cellulitis on the third or fourth day after surgery. The most frequent bacteria isolated from abscesses are staphylococcus aureus and albus.[10]

Loss of insulation or fracture of the catheter have also been reported and are rather frequent complications, due to increased angulation creating catheter breakage, usually at the level of the shoulder subject to the severe strain of frequent and vigorous motion of the arm and shoulders (Case 3).[1]

A coiling of the catheter electrode or actual rotation of the pulse generator may occur, with the extreme effect of a "fish reel" movement, in which the pacing catheter becomes wound around the unit dislodging it from its intracardiac position. This is, as a general rule, the result of discomfort over the pectoralis area, leading to rubbing over the incisional scar overlying the generator unit.[12]

Venous thrombosis has also been reported in association with permanent transvenous pacemaker catheters.[13] We have had the opportunity to observe one such case with venographic demonstration of thrombi involving the right external jugular, right jugular, right innominate vein, and partially superior vena cava extending to the right atrium. In our case, there was a venous obstruction in the right upper extremity and right side of the face. This complication at the extreme is seen when involvement of the right atrium and tricuspid valve leads to persistent chronic congestive heart failure. It is observed that the thrombi formations usually start at the site of a pacemaker catheter entrance in the vein, or from the site of contact of pacemaker and endocardium. This complication could lead later to pulmonary thromboembolus and, less frequently, to embolization to the systemic circulation if the foramen ovale is patent.[14-15]

In reviewing the literature, six cases of thrombosis associated with permanent transvenous pacemakers have been reported.[13-16] In only one case was the diagnosis made premortem. Our patient (Case 4) was treated with heparin and Coumadin, with good clinical response as a result of collateral formation.

Dermatitis, pulmonary air embolism, knotting of permanent electrode, and breakage with retention of catheter electrode have been added to the list of complications following permanent pacing.[17-20]

Summary

One hundred fifty consecutive cases of permanent transvenous pacemaker implants were reviewed for complications considered mechanical or non-electrical. Five cases were found, for an incidence of 3 per cent. These and other mechanical complications were discussed. Steps to avoid these complications as well as guidelines for diagnosis and management were also discussed.

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Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

Operant Conditioning Procedures

Conditioning in Schizophrenic Work

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EXPERIMENTAL research of behavior during the past 20 years has resulted in several very effective and reliable techniques for controlling behavior.[1] Recently, clinicians with greater interest in patient care than preserving antiquated theories, have been applying these techniques with a high level of success to the institutionalized patient.[2]

Behavior therapists view all behavior as learned behavior, with no distinction in this learning process being made between normal and abnormal behaviors. Some authors[3, 4] believe normal behavior and abnormal behavior to be reciprocally related, and the psychotic patient, therefore, exhibits very little normal behavior, but a considerable amount of abnormal behavior. Any action that will contribute to increase a psychotic's normal behavior repertoire will necessarily be beneficial to that patient. What follows is a demonstration of certain behavior techniques for the individualized treatment of one serious, inappropriate (abnormal) behavior, that of psychotic talk.

This study was a single subject design and was arranged to run for 30 sessions. The experimenter would verbally interact a maximum of 10 minutes each session with the subject if he continued working at his daily work task, and as long as he refrained from making seven inappropriate (psychotic) statements.

The purpose of this study was to determine whether or not attending types of stimuli would increase appropriate verbalization from the subject.

Procedure

Subject. The subject of this study was a 32-year-old white male, who has been an inpatient at the Veterans Administration Hospital in Topeka since 1961, with a diagnosis of schizophrenic reaction, undifferentiated type. He was accepted by the behavior modification unit in the later part of 1971, after failing to respond to the traditional methods used in the rest of the hospital.

The subject was chosen for this study mainly because he was quite verbal and would talk at length to anyone who would listen. Baseline data on the subject indicated that his statements were 70 per cent inappropriate.

Since his acceptance in the behavior modification unit, the patient has been "shaped-up" to a seven-hour a day, five-day work week. At the time of the study, the patient was working in the rug weaving program, finishing the rug by tying off the loose ends. This involves separating four strings, then using two separate 3-1, 3-1 cross-over slip knots. If the knots are not tied with equal pressure, the end of the rug can easily become unraveled, or be finished off unevenly. So, the task which is rather monotonous (and therefore rotated among patients frequently), demands a certain amount of manual dexterity.

The purpose of this therapeutic experiment was to increase the number of appropriate verbal statements in a psychotic patient while he worked, without interfering with his work productivity.

The amount of work productivity is easily measured by the amount of knots tied per hour. Prior to the beginning of this study, the subject was tying 12 knots per hour.

Method. The experimenter would seat himself across from the subject each working day and engage the subject in conversation. Beyond opening salutation by the experimenter, the tone and subject matter of the conversation were directed by the subject for each session.

During the initial appearance and upon disengagement of the interview, the experimenter would look at the patient directly, otherwise the experimenter would look only at the knots during the daily sessions. This was done to additionally focus the patient's attention on the task of tying knots and, thereby, enable the patient to earn tokens to pay for participation in activities of the unit. The patient was given initial instructions that he was to work at knot tying while they talked. No other reminder beyond this initial instruction was necessary throughout the study.

After seven inappropriate statements, the experimenter would get up and leave the work area for

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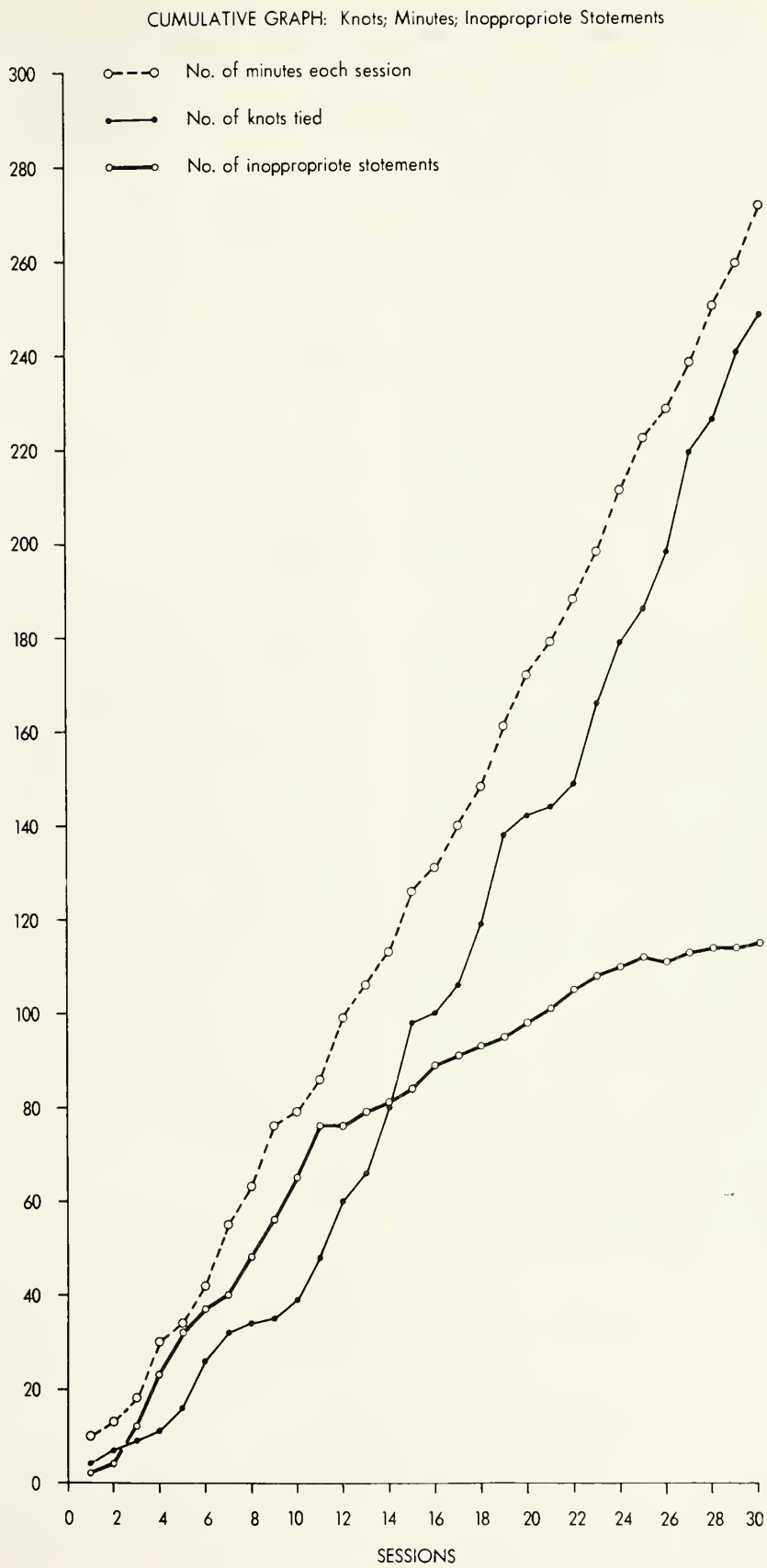
CUMULATIVE GRAPH: Knots; Minutes; Inoppropriote Stotements
No. of minutes eoch session
No. of knots tied
No. of inoppropriote stotements
300
280
260
240
220
200
180
160
140
120
100
80
60
40
20
0
0 2 4 6 8 10 12 14 16 18 20 22 24 26 28 30
SESSIONS

Figure 1

the day. To provide immediate feedback as to the appropriateness or inappropriateness of the conversation to the experimenter, a notebook system was employed in which a new page was used for each day. Two columns were apparent to the patient: Appropriate and Inappropriate. After each statement by the patient, a mark was made under the appropriate heading in the notebook. The patient was allowed, without penalty, to quickly glance at the notebook as each tally was made.

The experimenter would leave after ten minutes of appropriate conversation, or after seven inappropriate statements.

Results. The results indicate that the subject (1) increased his appropriate verbal responses; (2) decreased his inappropriate verbal responses; (3) increased and maintained maximum session time with the experimenter; and, (4) increased his work production level.

The cumulative graph *(Figure 1)* displays rather vividly how minutes spent with the experimenter and the number of knots tied increased proportionately, while, after an initial increase, inappropriate verbal responses decreased markedly.

Table 1 shows that session time and the number of knots tied correlate .77; session time and appropriate verbalization correlate .58; session time and inappropriate verbalization show a negative correlation of –.56; number of knots tied and appropriate verbalization correlate .79; number of knots tied and inappropriate verbalization also show a negative correlation of –.78, as does appropriate verbalization and inappropriate verbalization, –.56. All correlations are significant beyond the .01 level of significance.

Baseline data indicated the subject's rate of emitting inappropriate verbal responses at 70 per cent, with appropriate verbal responses at 30 per cent. During the study, the subject emitted 715 total verbal responses. From this total, it would be expected that approximately 500 (70%) would be inappropriate, and the remaining 215 (30%) would be appropriate. What was observed were 594 (83%) appropriate and 121 (17%) inappropriate responses.

Baseline data also indicated the subject tied 12 knots per hour. It would be expected, therefore, that in 3½ hours of total session time the subject would tie 42 knots. He actually tied 252 knots, which is six times the expected value.

TABLE I

	1	*2*	*3*	*4*
1. Session time		r = .77	r = .58	r = –.56
2. Knots tied			r = .79	r = –.78
3. Appropriate V.R. ..				r = –.56
4. Inappropriate V.R. .				

Table 2 indicates the results found between the expected and observed differences of appropriate and inappropriate verbal responses, and also the results of the difference found between the expected and the observed amount of knots tied, as shown by Chi Square.

TABLE II

	Appropriate	*Inappropriate*
Expected	215	500
Observed	594	121

$$\frac{(215 - 594)^2}{215} + \frac{(121 - 500)^2}{500} = X^2$$

$$X^2 = 955.38$$

$$P < .001$$

Discussion

The patient for this study was chosen because he emitted inappropriate verbal responses at a very high rate. Before he could be moved to the outpatient clinic, this rate had to decrease markedly and brought under stimulus control. Therefore, any experimental study that would attain this, would greatly enhance his probability of achieving that goal.

The study was designed not only to maximize the probability of decreasing verbal inappropriateness, but also, with special attention given, not to confound ongoing programs in which the subject was already engaged.

The study demonstrated that the subject's verbal inappropriateness decreased as his verbal appropriateness increased. It also demonstrated that the subject's work productivity increased substantially.

The results of this experiment corroborate prior studies which suggest that abnormal and normal behaviors are reciprocally related.

These findings lend themselves to further research in the clinical area.

Summary

An experimenter working in a one-to-one relationship with a psychotic inpatient substantially increased the patient's appropriate verbal responses by using operant techniques. Inappropriate verbal responses substantially decreased as the productive work level greatly increased in a total experimental time of 270 minutes spread over 30 daily sessions.

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(Continued on page 462)

Student THESIS

Sydenham's Chorea—Current Concepts and Literature Review

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PARACELSUS, in the Middle Ages, first applied the term "chorea" to the wild movements of religious fanatics visiting the healing shrine of St. Vitus. It was not until 1686, however, that Thomas Sydenham first described the childhood chorea which continues to bear his name. He attributed this disorder to the psychic trauma of emotional shocks and frightening experiences. In 1780, Stoll first postulated the association of this form of chorea with rheumatic fever. Since that time, his theory has increased in popularity, fostered especially by the work of Roger and Osler in the late 1800s. Thus it is that, despite many arguments and theories to the contrary, Sydenham's chorea remains a component of rheumatic fever—hence, its position is one of the major manifestations of rheumatic fever according to the Jones criteria. Unfortunately, however, this form of chorea remains rather poorly understood, and its treatment empirical. Therefore, it is the purpose of this paper to consolidate the information available on this disorder, and thereby, perhaps, make it easier to understand.

Etiology

The origin of Sydenham's chorea has been hotly debated over the years, especially with regard to its association with rheumatic fever.[1] The controversy is only further increased by reports of choreaform activity seen in such diverse disorders as Henoch's and Schönlein's purpura, systemic lupus erythematosis, postviral encephalopathies, hypocalcemia, hyperthyroidism, pregnancy, and many others.[2, 3] Whether this is true Sydenham's chorea, however, is debatable. Instead, the two most popular causes for this form of chorea continue to be (a) a preceding streptococcal infection; and (b) psychic trauma.

Proving that Sydenham's chorea is a manifestation of rheumatic fever, and hence a sequela of a streptococcal infection, has been a long and difficult task. As opposed to the other manifestations of rheumatic fever, which tend to appear up to five weeks after the streptococcal infection, chorea arises at a much later date. At that time, frequently, neither the memory of a "sore throat" nor laboratory evidence of a preceding streptococcal infection may be present. Extensive statistical studies, however, have shown that chorea, rheumatic polyarthritis, and carditis arise within the same patient with too great an incidence to be coincidental.[4] This, plus the concomitant occurrence of chorea, polyarthritis, and carditis has tended to stabilize its position as a manifestation of rheumatic fever. Just why a streptococcal infection should lead to chorea, arthritis, carditis, and the other components of rheumatic fever remains unknown.

While the infection theory remains the most popular cause for this form of chorea, the argument for psychic trauma as the cause has continued since Sydenham's time.[5] Citing the frequent lack of other signs of rheumatic fever and of laboratory evidence of a preceding streptococcal infection, many investi-

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This is one of a group of theses written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the School.

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gators have searched for evidence of emotional stress and instability or psychological problems in such patients. Such factors have been found often. Why the death of a loved one or the inability to express anger and anxiety should lead to chorea, however, has never been adequately explained.

Pathology and Pathophysiology

Little is known of the cerebral pathology in Sydenham's chorea, as patients only rarely succumb to this disorder. The changes that have been found are very similar to those seen in rheumatic fever alone. No consistent anatomical site is involved; lesions are found scattered throughout the cortex, basal ganglia, substantia nigra, and cerebellum. Basically, there exists an arteritis with cellular degeneration. Hyalinization of small vessel walls is seen, leading to their occlusion and subsequent infarction of surrounding tissue. Perivascular inflammation and petechial hemorrhages are also present. Cellular damage consists of nuclear and cytoplasmic degeneration, and may progress to complete destruction of cells.

The pathophysiology of Sydenham's chorea is unknown. In theory, there is a release of inhibitory mechanisms from extrapyramidal or cerebellar sources. This is associated with reappearance of the "H" reflex—a monosynaptic stretch reflex normally lost at age six months but reappearing in chorea. Electroencephalograms frequently show a diffuse dysrhythmia with slow waves and 4-6 cycles per second (cps) paroxysmal activity.

Clinical Characteristics

Sydenham's chorea occurs most often between the ages of five and fifteen, with a female to male ratio of 2:1. It may arise from two to six months after a preceding streptococcal infection, usually a pharyngitis, and can be present with or without the other manifestations of rheumatic fever. If occurring with the latter, chorea is seen most often with a subclinical carditis, and only rarely with arthritis.

This disorder usually begins insidiously, with the patient experiencing increasing irritability and restlessness and becoming more emotionally labile. Gradually, there is the onset of the three most outstanding clinical features: spontaneous movements, incoordination, and weakness. The movements are abrupt, brief, nonrhythmical, and can involve any portion of the body. They may begin on one side and remain unilateral (hemichorea), or begin and remain bilateral. Such movements tend to occur even at rest, but tend to disappear during sleep. Incoordination becomes apparent due to the interruption of voluntary movements by the spontaneously interfering movements.

Clinical findings characteristically present in chorea include the following:

1. Inability to maintain contraction of the hand when grasping the examiner's fingers (relapsing, or "milkmaid's grip").
2. Inability to maintain protrusion of the tongue without closing the jaw.
3. A tendency to pronate one or both hands when extending the arms over the head.
4. Hyperextension of the fingers and hands when extending the arms in front.
5. "Hung-up" deep tendon reflexes.
6. Diffuse hypotonia.
7. Speech which is indistinct, jerky, and explosive, perhaps associated with irregular breathing.
8. A generalized restlessness and emotional lability, coupled occasionally with inappropriate behavior.
9. Deterioration in handwriting.

Neurologic deficits and pathological reflexes are occasionally seen in Sydenham's chorea.

Laboratory Determinations

Unfortunately, there are no laboratory determinations which are especially useful in the diagnosis of Sydenham's chorea. Even the acute phase reactants and streptococcal antibody titers, so useful in diagnosing rheumatic fever, are nonspecific. The erythrocyte sedimentation rate is normal in 83 per cent of patients with chorea only; in 41 per cent of patients with chorea and other rheumatic manifestations; and only rarely in rheumatic fever alone. Likewise, the C-reactive protein is negative in over 80 per cent in the first group; 45 per cent in the second; and only rarely in the third. The streptococcal antibody titers are not much better in the diagnosis of Sydenham's chorea. The anti-streptolysin (ASO) titer is much greater in pure rheumatic fever than in pure chorea, averaging 360 units/cc. versus 180 units/cc.[6] Ninety-five per cent of patients with rheumatic fever without chorea studied within one month of onset had ASO titers greater than 200 units/cc; 73 per cent of patients with chorea only were at this level at one month from onset, with this figure decreasing to 45 per cent later in the course.[4, 7] Antihyaluronidase titers show the difference between rheumatic fever with chorea and chorea alone: 380 units/cc versus 98 units/cc.[6]

Differential Diagnosis

Tics, the quick, repetitive, patterned movements usually involving the face, are most often confused with mild chorea. Differentiation is possible by observation that these movements are available to the patient's consciousness, can be altered at will, and do not interfere with coordination. The movements

of Wilson's disease (hepatolenticular degeneration) may simulate Sydenham's chorea, but when present they are usually associated with a Kayser-Fleischer ring. Huntington's chorea may occasionally arise in childhood, but can be differentiated by family history. Choreo-athetotic cerebral palsy can be separated only by careful observation.

Treatment

Many modes of treatment have been tried to relieve the abnormal movements of Sydenham's chorea. In the past, arsenic, calcium, vitamins, Lugol's solution, antisera, intrathecal magnesium sulfate, and others have been used. Hyperthermia as high as 106 F (41.1 C), generated by typhoid vaccine, Nirvanol, or applied externally, has been shown to decrease the choreiform movement; fatalities from such treatment have occurred, and thus, it is no longer in use. Corticosteroids and salicylates, as used in rheumatic fever, are without benefit.

Present treatment primarily involves sedation with phenobarbital or chlorpromazine, as the choreiform movements have been observed to increase with stress and decrease with relaxation and sleep. An environment free from noise and bright lights is essential to instill in the patient a sense of quiet and calm. The environment should also be structured so as to protect the patient from self-injury; bed rest and limited activity should be continued until the patient is better able to control the movements.

A recent advancement in the treatment of chorea of any origin is the use of haloperidol 2-6 mg/day (0.1 mg/kg/day). This butyrophenone has a mechanism of action very similar to the phenothiazines, but is more potent than the latter. Both haloperidol and the phenothiazines are known to increase the turnover of dopamine in the corpus straitum by blocking dopamine receptors. A drug-induced parkinsonism often results from administration of these drugs. In contrast, the administration of dopamine can lead to choreiform activity and other extrapyramidal side effects. Hence, it seems reasonable to treat a condition which may be related to dopamine by a drug shown to block dopamine receptors. Further controlled studies are necessary to prove the usefulness of haloperidol, however.

Prognosis

Most commonly, the manifestations of Sydenham's chorea tend to fade with time—the intervals being from a few months to as long as two years. Sequelae to this disorder do exist, however. As many as 33 per cent of patients not treated with prophylactic penicillin may go on to develop rheumatic heart disease with time, this being more common in patients initially seen with chorea and other signs of rheumatic fever than in those with chorea alone.[3] Studies have shown that the mortality from rheumatic heart disease 20 years after onset is about 60 per cent in patients with chorea and other rheumatic manifestations, and only 12 per cent in those with chorea alone. Many neurologic and psychological sequelae are mentioned in literature, including convulsions, hyperkinesis, a decreased learning ability, schizoid symptomatology, behavioral problems, and others.[7]

As is possible with rheumatic fever, recurrences of Sydenham's chorea do occur.[8] Essentially the same course is followed as in the initial disorder. It is for this reason that long-term antibiotic prophylaxis should be carried out for those patients with Sydenham's chorea as is for those with the other manifestations of rheumatic fever.

Summary

Sydenham's chorea, perhaps the rarest manifestation of rheumatic fever, is a poorly understood and empirically treated childhood disorder. It occurs most often in girls aged 5-15, who have experienced a streptococcal infection some 2-6 months previously. The characteristic clinical features begin insidiously and consist of abnormal spontaneous movements, speech difficulties, incoordination, and weakness. Symptoms may persist for as long as 6-12 months. Laboratory tests, such as streptococcal antibody titers and sedimentation rates, are less helpful in the diagnosis of Sydenham's chorea as opposed to their value in diagnosing acute rheumatic fever with carditis. Hence, to diagnose this disorder one must rely primarily on clinical signs and symptoms. Treatment presently consists of sedation (phenobarbital or chlorpromazine) and rest, until the patient becomes able to better control the movements; salycilates and corticosteroids are of little value in treatment. The current use of haloperidol in Sydenham's chorea appears promising, but more investigation remains to be done. Even with treatment, however, sequelae to this disorder do exist, including rheumatic heart disease and neurologic and psychologic disorders. Recurrences of the choreiform activity may occur, particularly after medications referred to above are withdrawn.

It is for this reason, and since approximately one third of patients not treated with monthly parenteral penicillin injections may develop rheumatic sequela, that antibiotic prophylaxis is needed in Sydenham's chorea, as with the other manifestations of rheumatic fever.

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NEW LEARNING SYSTEM ON CVD

A new learning system about cerebral vascular disease, now available to physicians or physician groups has been announced by the pharmaceutical company of Marion Laboratories, Inc. The learning system consists of a color film, "Diagnosis and Treatment of Cerebral Vascular Insufficiency," together with a supply of monographs that accompany the film. The monograph serves as a convenient review of information in the film and contains a quiz to be self-administered.

The film is suitable for continuing education credit hours from the American Academy of Family Physicians and the American Medical Association. It discusses the prognostic value of the various indications in the stroke-prone profile; describes the work-up of the patient diagnosis in cerebrovascular disease, including the use of cerebral angiography; describes the categories in the differential diagnosis of cerebrovascular disease; explores current modes of therapy in the management of acute thrombotic stroke; describes medical therapy, including the use of anticoagulants, platelet inhibitors, and glycerol; and discusses surgical treatment.

The editorial committee for the film is: Clark H. Millikan, M.D.; Michael E. DeBakey, M.D.; Lawrence C. McHenry, Jr., M.D., and John Stirling Meyer, M.D.

Physicians may obtain the film without charge for viewing themselves or for showing to physician groups by writing: Professional Services, Pharmaceutical Division, Marion Laboratories, Inc., P. O. Box 9627, Kansas City, Missouri 64134.

Human Mycoplasma Infection

(Continued from page 444)

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Operant Conditioning Procedures

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Socio-ECONOMICS

Supplemental Security Income Program

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THE NATIONWIDE program of direct federal payments to aged, blind, or disabled persons with limited income and resources goes into effect on January 1, 1974. Known as Supplemental Security Income (SSI), the new program will have uniform eligibility requirements for such persons to replace the multiplicity of requirements existing under the present federal-state public assistance programs.

The Supplemental Security Income program will be wholly financed from federal general tax revenues. Responsibility for administering the program has been given to the Social Security Administration (SSA) not only because of their experience in managing a monthly benefit payment program and the existing SSA advanced data processing system, but also because of the well-established nationwide network of SSA offices and program centers.

The title of the program—Supplemental Security Income—indicates that these benefits are expected in most cases to *supplement* income from other sources, including Social Security benefits. Those persons receiving public assistance on the basis of age (65), blindness, or disability according to state plans in effect for October 1972, and who received such aid for December 1973, will, in general, be converted to the federal rolls beginning January 1974. Further, blind and disabled recipients will continue to be considered blind or disabled for SSI program purposes so long as they continue to meet the definition of blindness or disability under the state plan or the provisions for blindness or disability that apply to new claimants under the federally administered program after December 1973. According to preliminary data, it is estimated that about 6.2 million people including approximately 1.6 million blind and disabled people will be eligible in January 1974 on this basis.

The federal law will pay many people who are not now eligible under state programs because they have income or resources above specified levels, or because their states have requirements making relatives responsible for their care. Also, many people who actually meet the state requirements do not apply for public assistance payments in states which have lien laws. Since the federal law has neither lien law nor relative-responsibility provisions, more people are expected to apply.

The states may, at their own option, elect to supplement the federal SSI payment. Estimates are that about a million of the SSI recipients will receive additional state aid beyond the federal payment.

The SSI program will generally use the same definitions of disability and blindness used in the social security disability insurance program for determining eligibility in new claims. To help simplify and speed the processing of disability decisions and to insure uniform treatment of all applicants, no matter where they live, the medical evaluation criteria developed for the Title II disability insurance program (Social Security) with the aid of practicing physicians, medical organizations, and the medical advisory committee to the Social Security Administration have been generally adopted for the SSI program. The evaluation criteria describe in terms of symptoms, signs and laboratory findings, impairments that reflect the level of severity that would prevent most people from working for a year or longer. These criteria are constantly being refined to reflect advances in medicine and to take into account disability program experience.

If an applicant has an impairment or a combination of impairments that meets or equals the criteria, and he is not working, he would generally be considered disabled. Most allowances are based on medical considerations alone—that is, the claimant's impairment meets or equals the level of medical severity in the criteria. It is also possible for an impairment to be slight or minimal, thereby resulting in a denial strictly on a medical basis. However, for workers

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who have impairments which fall short of the listed level of severity but which prevent them from doing their previous or customary work, consideration is given to their ability to do any other work in light of their remaining capacity and of their age, education, training, and work experience. In these cases, the individual must not only have an impairment which prevents him from doing his usual work, or work he has done previously, but also must be unable to do other kinds of work for which he is reasonably suited. In the situation where an older worker with a marginal education and a long history of arduous unskilled physical labor has an impairment which prevents him from doing his usual work, he may be considered under a disability.

All persons whose applications for determinations of disability are adjudicated in a state disability determinations unit are referred to the state vocational rehabilitation agency for consideration of rehabilitation services. The states will be fully reimbursed by the federal government through the Rehabilitation Services Administration for the services they provide to qualified disabled and blind SSI recipients.

With the anticipated doubling of the state disability determinations unit workloads, emphasis will be placed on expanding resources within the medical community so that it will be possible to get medical reports needed for adjudication of claims as quickly as possible. Although generally the same guides apply under Title II and Title XVI, there are some differences. For example:

1. *No Waiting Period*—Under Title XVI (SSI), an individual who is determined to be blind or disabled will be eligible for payment for the first month in which he has filed an application and is disabled. There is no set waiting period which must be served after the onset of disability and during which payment cannot be made. (Under Title II, a five-month waiting period must be served after the onset of disability.)

2. *Presumptive Disability*—The law provides that an applicant for disability benefits who is found to be "presumptively disabled" may be paid, under certain conditions, for as many as three months while a formal determination of his disability is being made. This provision, along with the absence of a waiting period under SSI, will intensify the need for obtaining medical evidence more rapidly, so that disability determinations can be made promptly on claims filed by needy SSI applicants.

3. *Childhood Disability*—With the implementation of the SSI program, the Social Security Administration will for the first time be responsible for disability evaluation and payment for children who are under the age of 18. A child of a family with limited income and resources will be found disabled if the child has a medically determinable physical or mental impairment which can be expected to result in death, or which has lasted or can be expected to last for at least 12 consecutive months and is of comparable severity to that which would prevent an adult from engaging in substantial gainful activity. The question of vocational assessment and concomitant ability to engage in substantial gainful activity is generally not relevant in evaluating disability during childhood because, in most situations, the child will not be of an age where he could reasonably be expected to enter the working population. Thus, in childhood cases, a finding of disability will be made solely on the basis of medical considerations including special medical criteria being developed for these cases within the above framework of consideration. There are, for example, severe impairments unique to childhood cases which are not now specifically described in the Social Security Listing of Impairments. The new medical criteria with appropriate signs, symptoms, and laboratory findings are being formulated to evaluate these cases. There will also be a need for frequent pediatric reports under the new program. Similarly, there may be a need for the Kansas state disability determinations unit to have pediatricians on its staff or at least available for consultation to review reports on these types of cases.

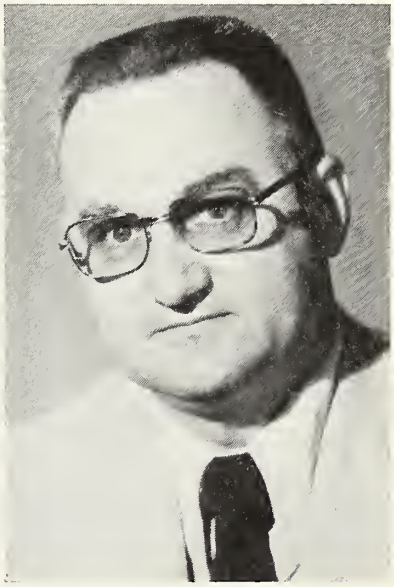
4. *Drug Addiction and Alcoholism*—The law provides that a disabled person, who has also been medically determined to be a drug addict or alcoholic, shall be eligible for SSI payments only if he is undergoing treatment appropriate for his condition as an addict or alcoholic at an approved institution or facility, if one is available. An eligible individual who has been medically determined to be a drug addict or alcoholic must receive benefits via a representative payee.

5. *Blindness*—The criteria for establishing blindness under SSI are identical to those required to establish statutory blindness under the Social Security disability insurance program. Unlike Title II, however, engagement in substantial gainful activity will not preclude SSI payments if the statutory definition of blindness is met, although the SSI payments may be reduced under the income test. Also, since there is no duration requirement for blindness under SSI, there can be a favorable decision based on temporary blindness. Once again, the need for comprehensive and prompt medical reports must be underscored.

Implementation of the SSI program will undoubtedly give rise to new questions and point out areas of concern with respect to the medical community and the state agencies. If you have any further questions or desire additional information, please contact the author at Box 5057, Topeka, Kansas 66605, or telephone collect, 1-800-432-2428.

The President's Message

What Are You Worth?



About five or six years ago, I attended a meeting in Ann Arbor, Michigan, and during part of this meeting, I attended a lecture on cost benefits as applied to health care, using an analytical approach. The speaker outlined ways in which costs are determined by industry, and controls by which these costs can be maintained by the industry, and then applied this cost benefit analysis concept to the health care industry. I was disturbed at the time. I felt that people are taken care of by physicians, that they are individual people, and that they have individual problems. It seemed to me that using the method of cost benefit analysis would depersonalize people and dehumanize them. I really did not feel that one could apply the concepts of cost benefit analysis to individual people and still maintain their status as a human being and as a person.

Subsequent events have seemed to indicate that our federal government, through Congress and the bureaucracy of HEW and Social Security, intends on using cost benefit analysis as the method by which cost of medical care will be controlled. We have already in 1973 been exposed to letters sent out by high government officials in which institutional care will be controlled by a theory known as Total Hospital Expenditure. This concept states that if a hospital collects, for example, one million dollars in receipts in one year, it will, during the following year, be allowed to collect one million dollars plus a given percentage. This will stifle progress of medicine. It will make it impossible for us to use any type of new approach to medical care. It will impose upon us the mandatory requirements of utilizing less expensive methods of treatment (which may not be as adequate as the more expensive methods).

I am convinced that there are basically two parts to the Professional Standards Review Organization: one deals with cost containment; the other deals with the quality of medical care. The Kansas Medical Society believes in quality of medical care to our people in Kansas. I do believe that the people who will administer Professional Standards Review Organization are interested primarily in cost containment, and that the methods as described for usage in cost benefit analysis will be utilized for that cost containment.

I would like to suggest to you that if you have two patients, both of whom need some type of major diagnostic or surgical procedures done, each of which cost, for example, $4,000, and if under the cost benefit analysis theory you have but $4,000 to spend, which patient are you doing to spend it on?

WHAT ARE YOU WORTH—AND TO WHOM?

Best Wishes for the Holiday Season!

Thomas F. Taylor

President

Dilemma du jour

As world population approaches the saturation point, there is increasing concern about the quality of human life as well as the quantity, and the desirability of maintaining life is coming increasingly under scrutiny. True, through all times there have been those who viewed life with a jaundiced eye and saw no reason to retard those natural threats to life, either individual or collective. But the admonition to go forth and multiply has been in effect for a good many centuries, and the fact that it now seems imperative to modify it is testimony to its success. This plus an increasing public concern with medical matters and an increasing public insistence on participating in the establishment of new policies have made the question of the maintenance of life one of the day's more popular subjects for debate.

The area involved is extensive. The care of aging has come to the fore, and it is impossible to consider it in any depth without some examination of the extent to which the maintenance of life is justified. But age alone is not the focal point. Trauma and illness in young people may present the same problem with somewhat different emphasis. Individuals totally incapacitated both in effect and potential survive through the nominally compassionate ministrations of the professional attitude that we must apply all available ability to the fulfillment of the letter of our obligation. There is still a sense that anything short of this maximum effort to maintain life is tantamount to a betrayal of this obligation and becomes a willful imposition of death.

Society has always had a prejudice against the taking of human life except in certain circumstances —the defense of one's self, the punitive retaliation of society against a major crime or, on a larger scale, as an expression of frustration when words are no longer effective communication between governments. Since the taking of life (even one's own) is abhorrent, the decision to allow a person to die must overcome a guilt-by-association.

Euthanasia is the term usually applied to the policy of permitting death to intervene earlier than capabilities and facilities would require, but there is already some question as to whether it is adequate or even means the same thing to all people. The derivative definition barely suggests the complex of social and medical elements which come under discussion in a world capable of manipulating vital processes almost at will.

Early in the discussion, it is popular to disclaim any interest in actively promoting death. The collective moral intellect can scarcely contend with more than the concept of permissive death as it strives to establish criteria and guidelines. Perhaps the difference is simply quantitative—with more subjects to decide about, we may one day accept more easily the idea of active (board-certified?) termination. Perhaps it is temporal—we have reached the point in time when the milder concept can be spoken of with impunity, and we have only to wait a few more years to speak of an accepted, willful imposition of death.

Why is the problem so difficult for the physician? It can be said the fundamental principle of his calling is the defense of life and delay of death to the ultimate point. It may be only a matter of time, then, until the medical philosophers decide that the real service of the profession is not the obsessive battle with death but the welfare of the patient, and that welfare is not always synonymous with the retention of life. It will probably require not so much a profound change in medical philosophy as a change in the social philosophy which persists in considering death abhorrent. The physician's exposure to the terminal patient, his experience and compassion, would more often bring him to the decision for action (or inaction) were he not the product of and limited by a social order that rejects such an attitude.

But his experience may be the thing which makes it more difficult to determine the time and the propriety of the decision. Most discussions about the termination of the life of a hopelessly ill person introduce the matter fully developed, pose the question with the patient already in extremis. This is not the way the physician experiences it. He offers his first advice and direction generally far in advance of this situation. As the patient progresses in his course, the physician adds to, discontinues, revises his advice and therapy—but it is a continuing process. At what point does he say, "This far and no farther"? In the popular idiom of the day (and with the specious

validity of such terms), this is known as "pulling the plug." This indicates, of course, that the earlier decision was made to insert the plug in the first place. The physician must determine at what point his ally, the machine, becomes the enemy. The point of decision for the physician is the acceptance of the fact that his effort may have been good but it is now fruitless.

Neither the physician nor society can pursue the problem far without considering the influence of the identity or personality of the patient—and this is true not only in the death decision. Genetic counseling experience has disclosed that families faced with the possibility of producing a defective offspring will accept the risk more readily if the condition will result in early death of the child (before identity is firmly established?), than if the prospect is less ominous but carries the risk of long and burdensome maintenance of an incapacitated child.

A discussion of the termination of life is simplified if it is focused on the hypothetical case. This avoids any need to consider the personalities of the subject, physician or family, for these exist and are manipulated only in the concept of the discussion. But in the actual case, neither physician nor family can be free from such effect. Whatever the direction of his advice or decision, the physician cannot be free of the identity of his patient—or his own. The highest virtue of the concept of the Living Will, in which the individual, well before the fact, requests a sensible, non-emotional limit to life-sustaining efforts in his behalf, is that it introduces the personality of the individual into the decision and relieves the family and physician of some measure of guilt and societal criticism which might obtain without it.

Every rule man lives by, whether statutory, religious, moral or personal, exists first as a thought, a sense of need. It becomes defined and recorded either as law, as tradition, as social dictate, or personal attitude. But up to this point, it is theory. The viability of a rule depends upon its practical usage. The maintenance of life has been supported by religion, law, mores and personal desire, but now society has increased in physical quantity, collective emotional maturity (hopefully), and rational interpretation to the point that it must face the problem of the desirability of life. And not just in the individual instance in which a physician may assume to himself the making of the decision or acquiesce to the dictates of others if they are counter to his own feelings.

Some physicians argue that only by exhaustive use of all available life preserving techniques can we continue our steady improvement of the health of mankind. They remind us that the inexplicable is routine in medicine and ask: what of those who have been abandoned only to survive by some inner capacity unrecognized by ordinary standards? Others will claim that the higher professional goal is the welfare of each individual, which necessitates at some point the acceptance of the inevitable. For the physician at large, this is the crux of the problem—the reconciling of his obligation to sustain life with his obligation to serve the welfare of the patient. If one wishes to play God, it is essential to know what God intends. But it is just as necessary to know this if one wishes *not* to play God.—*D.E.G.*

PSRO

Guidelines on how organizations can apply to become PSROs within the designated areas will be issued in February. By next June, HEW hopes to have 50 PSRO's chosen. Within four to six weeks, a PSRO bulletin will be sent to all physicians in the nation outlining the status of the program and informing them of PSRO developments.

It is predicted that from 20 to 30 small states will be single-state PSRO areas.

Prior to the HEW decision against statewide PSROs in large states, the Senate Finance Committee had tentatively approved a provision that would ban HEW from using a limitation on the number of physicians that may belong to a PSRO. If enacted, the provision could make it easier for statewide PSROs to win HEW approval.

At present, the Department is employing a general top limit of 2,500 physicians per review organization, a maximum guide that obviously would foreclose larger states from having a single organization to review institutional care for Medicare and Medicaid patients.

The amendment was sponsored by Sen. Lloyd Bentsen (D.-Texas) and agreed to by Sen. Wallace Bennett (R.-Utah), originator of the PSRO concept and a staunch proponent of smaller PSRO units.

The language of the proposed Bentsen amendment reads: "In carrying out the provisions of this section, the Secretary may designate, as an appropriate area with respect to which a Professional Standards Review Organization may be designated, an area encompassing a whole state; and the Secretary shall not refuse to designate any qualified organization as the Professional Standards Review Organization with respect to such area solely because of the number of physicians participating in such organization."

Whether or not the Senate committee action on a House-passed measure making technical changes in the Social Security Law would result in a significant change in HEW PSRO policy is not known at this time.

KMS Obituaries

MARCELLA M. KRAHENBUHL, M.D.

Dr. Marcella M. Krahenbuhl, 51, of Shawnee Mission, died September 29, 1973. She was born in Kansas City, Missouri, on June 28, 1922.

Dr. Krahenbuhl was graduated from the Washington University School of Medicine in 1950. She had practiced in Kansas since 1954.

WILLARD C. SCHWARTZ, M.D.

Dr. Willard C. Schwartz, 73, of Manhattan, died November 18, 1973. He was born March 18, 1900, in Hutchinson.

Dr. Schwartz was graduated from the Washington University School of Medicine in 1927. He established his practice in Manhattan in 1931, where he practiced until his retirement in 1971.

Survivors include two daughters and two sons. A memorial fund has been established with the KU Endowment Association.

ALBERT E. TITUS, M.D.

Dr. Albert E. Titus, 88, of Cottonwood Falls, died November 1, 1973. He was born October 25, 1885, in Shawnee County.

Dr. Titus was graduated from the Kansas Medical College, Topeka, in 1912. He practiced at the same location for 60 years.

Survivors include a daughter. A memorial fund has been established.

HAROLD O. WILLIAMS, M.D.

Dr. Harold O. Williams, 71, of Cheney, died October 3, 1973. He was born January 19, 1902, in Pleasant View, Missouri.

Dr. Williams was graduated from the St. Louis University School of Medicine in 1929. He was licensed in Kansas the same year, where he remained for the rest of his medical career.

Surviving Dr. Williams is his wife.

Woman's Auxiliary

The Master Key

The spirit of Christmas is in the air, and we feel a deep sense of gratitude and appreciation for the opportunities we have as doctors' wives, for the means of serving in the community, thus helping you in any way we may. Yes, our love and belief in *you* are behind a great deal of the effort we, your wives, make in service to individuals and the community, this together with our faith in the Almighty, and the benefits of living in this good land!

It is true, some wives do not participate in community affairs, but the many who do are motivated by the best of all possible incentives—to serve. This is a reflection of YOU, and your own service and concern. Yes, *you,* and the medical society to which you belong, provide us the Master Key. You have been an example to us, and it seems altogether appropriate that we try to make some contributions too. Many times, these are in areas you simply don't have time to cover, but it is only with your approval and guidance that we assume new undertakings.

Please let us dispel the idea that Auxiliary has to come first. No, indeed; our first obligation should always be to our doctor-husbands, our families, and the home. The Auxiliary does not compete with any of these for time or the attention given by your wives. We leave priorities up to them; ours is a truly democratic organization. But we do ask that Auxilians save *some* of their time and energy for our mutual undertakings, as we feel we are in a situation to give form and organization to educational and service-oriented programs and plans.

We hope that you feel the Auxiliary is important too; that you recognize the unique position we have in the community; and that as educated women we are in a position to help you by (1) educating ourselves to community need; (2) serving then as catalysts and opinion makers; (3) setting up and leading in volunteer programs for education and service; (4) calling attention to the doctors' first interest in providing the best medical care possible . . . all in your name, and for the community.

So, please suggest to your wife, if she doesn't belong, that she join her local medical Auxiliary. She'll be doing you, herself, and all of us a favor. We need her; she needs us. For "We have the keys to a better Understanding of Health"; we want her to help us use them, and we need our Master Key most of all!

Katie Keys

115th Annual Session

KANSAS MEDICAL SOCIETY

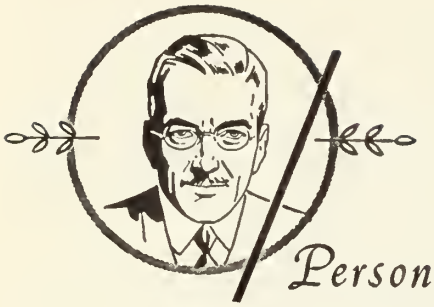
May 5-8, 1974, Topeka

MODERN ASPECTS OF TRAUMA

1. **Aeromedical Transportation of the Injured**
2. **Medico-Legal Aspects of Trauma**
3. **Symposium on Fat Embolism**

The 1974 meeting will be held in conjunction with the following specialty societies:

KANSAS SOCIETY OF PATHOLOGISTS
KANSAS OBSTETRICAL AND GYNECOLOGICAL SOCIETY
KANSAS ALLERGY SOCIETY
KANSAS SECTION ON EAR, NOSE AND THROAT
KANSAS SOCIETY OF ANESTHESIOLOGISTS



Personalities—IN KANSAS MEDICINE

Duane K. McCarter and **William B. Triplett**, both of Topeka, participated in a continuing education workshop on respiratory problems and treatments conducted by the Shawnee County LPN Association.

Among Fellows newly installed into the American Academy of Family Physicians were **William T. Read**, Coffeyville; **G. George Ens** and **Robert G. Franz**, Hillsboro; **A. K. Ratzlaff**, Goessel; **T. C. Ensey**, Marion; **Kenneth McLain**, Ransom.

Ernest W. Crow, Wichita, directed the two-week course on coronary care, sponsored by the Heart Association.

"Lung Disease in My Practice," was the topic of discussion for **Yong W. Kim**, Concordia, at a conference concerning the care of lung disease patients.

Rex R. Fischer, Manhattan, spoke for the UFM Doctor's Series on the topic of "The Women's Liberation Effect on Modern Medicine."

Kansas Heart Association, Inc. installed **Major M. Swan**, Great Bend, as its new president, succeeding **William J. Von Ruden**, Hutchinson.

John Cody, Hays, appeared on Channel 12 cable television show, "Let's Talk," recently.

Among those attending the American College of Surgeons annual meeting in Chicago was **Harl H. Stump**, Hays.

Harvey J. Meulbroek, Wichita, participated in a series of discussions held in Hesston on the topic of death and dying.

"Family, Kids and Community," was the general topic for discussion in which **V. W. Steinkruger**, Phillipsburg, participated.

D. Cramer Reed, Wichita, was recently named Vice President for Health Education at WSU. He served as Dean of the College of Health Related Professions from 1970 until this fall.

Addressing the Hillsboro Lions Club recently was **Robert H. Kelly**, Augusta.

Marvin R. Gunn, Salina, was the speaker at the regular meeting of the local Medical Assistants society.

Participating in a public meeting as a speaker on arthritis was **E. Dean Bray**, Minneapolis.

Ramon W. Schmidt, Garden City, was the speaker at a recent annual meeting sponsored by senior football players' parents. His presentation dealt with the protection of health and marathon running.

Chairing the council planning committee for a camporee at the Cheyenne Scoutlands, designed to promote physical fitness competition, was **Robert C. Polson**, Great Bend.

Charles H. Miller, Parsons, was cited by the Business and Professional Women's Club as the citizen of the year.

The D. C. Line

Editor's Note: The Editorial Board feels that the Kansas Medical Society would profit by maintaining contact with Representative Bill Roy, both as a source and interpreter of political activities on the national medical front. Accordingly, we are initiating a monthly column written by Dr. Roy. It is hoped that this will not only provide the service intended, but invite a response of readership either through the JOURNAL or to Dr. Roy directly.

In my opinion, there is no better time than now to pass national health legislation. One reason is that important present laws, one providing federal dollars for training health manpower and the other supporting comprehensive health planning, will expire June 30, 1974. This legislation will be evaluated and then permitted to expire, or will be re-enacted or replaced by new legislation.

There are, I believe, at least four other reasons to enact necessary legislation now.

First, Americans are now paying a great deal for health care—$84 billion last year and an estimated $105 billion next year—and while concern is rapidly mounting, the people are not yet in open rebellion about health care costs. But costs are rising so rapidly that more and more people are being priced out of the health care market each day. Health care expenditures are increasing $23 million per day. More and more people are saying to their elected representatives, "Do something."

If something is not done now, the outcry will reach a crescendo, and then we, who are elected representatives, will respond, probably quickly and probably unwisely.

We have not yet reached the public demand for action now. There is still time to act in a considered, incremental, and evolutionary way.

Secondly, there is today among physicians a great awareness of existing problems, a great reservoir of good will, and a great deal of innovative, constructive activity. Day after day, practicing physicians come by my office to tell me of their efforts to improve health care delivery. Physicians, other health professionals, private and public hospitals, private health insurance companies, and innumerable others would like to join with government in meeting the health care needs of the American people.

There is no guarantee that this willingness to work together will continue. There are areas of both real and apparent conflicts of interest among health interest groups. But for now, this spirit of cooperation exists.

A third good reason for the consideration of health care legislation now is that the overwhelming majority of the members of Congress at this time do not want to interject massive federal programs into the health care delivery system. Most members of Congress are aware and somewhat intimidated by the size and complexity of the health care delivery system, and most present members of Congress date back far enough to have been burnt by the erroneous actuarial estimates for Medicaid and Medicare. I find that my colleagues in Congress, as a whole, would very much prefer that the medical profession and other health professionals solve most health care delivery problems, or alternatively, that we, the health professionals, propose and support the minimal legislation that will work to meet today's health care problems. However, most members are aware that something must be done, reasonably soon.

The Administration in power has to be considered a fourth major factor in determining whether this is a propitious time to pass major health care legislation.

I will discuss the "Administration factor" in a future column.—*W.R.R.*

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

Official Proceedings

1973 Fall Meeting of the House of Delegates

The Fall Session of the House of Delegates of the Kansas Medical Society was instituted principally to work on matters of legislative interest and for such emergency resolutions as might appear.

The resolutions acted upon by this House of Delegates are printed in numerical order under the minutes of the Second Session. All other resolutions introduced were delayed for action until May.

FIRST SESSION

The first meeting of the House of Delegates was called to order by the Speaker, Dr. Clair C. Conard, at 10:00 a.m. on Sunday, November 18, 1973, at the University Ramada Inn, Manhattan. He announced the composition of the House of Delegates as follows: representing component societies, 139; councilors, 18; officers, 7; past presidents, 21; representing specialty societies, 15; for a total of 200.

Dr. Thomas F. Taylor, President, introduced the Honorable Robert Docking, Governor of Kansas, who spoke concerning the Kansas Foundation for Medical Care, KUMC, and HMO.

Dr. John N. Blank announced the forthcoming Leadership Conference, to be held at Crown Center, Kansas City, on February 1-3, 1974. All physicians were being encouraged to attend this meeting.

Dr. Conard next introduced Mr. Dave Morrison, the AMA Field Representative, and Mr. Hank Parkinson, Wichita, the director of public relations for the Kansas Medical Society.

The next speaker was Dr. Dwight Lawson, Topeka, the State Medical Director for the Social Security Disability Determinations Unit. He presented a report on Title XVI, the Supplemental Security Income Program which will go into effect on January 1, 1974.

Dr. Taylor reported on his travels over Kansas, in conjunction with the Council district meetings.

Dr. Francis T. Collins, President of the Kansas Foundation for Medical Care, presented the following report:

Kansas Foundation for Medical Care

Because of the uncertainty, confusion, half-truths and misunderstandings about PSRO, and because of my involvement and experience with some implications of the current PSRO legislation, I feel it is my duty to try to give the most complete, factual, and current information concerning PSRO and the Kansas Foundation for Medical Care that I can. This is my evaluation of where we are in Kansas at the present time, relative to federal legislation and PSRO.

The facts as we know them at this time

PSRO has been signed into law and goes into effect January 1, 1974. It is a poor law in many respects, but we are going to have to live with it, or some modification of it. The main difference in opinion between organized medicine and congressional views is that Congress sees this as a cost-control measure primarily. Organized medicine sees it as a quality control and a help to try to control the escalation of medical costs. The details have not been finalized because of the lack of definitions and the lack of bureaucratic interpretation.

Presently, the law will apply to all federally funded in-hospital services and in stage I nursing homes, relative to necessity for admission, length of stay, conformity to quality of services, and location of level of care.

It is implied in the law that after January 1, 1976, the program will be expanded to home and office care and other nursing homes categories.

Until January 1, 1976, only organizations of physicians or groups representing the majority of physician-providers in an area can be designated as the PSRO agent. This designation is on a temporary basis and is renewable annually. By January 1, 1976, if the function of the present PSRO is unsatisfactory to HEW, or the original group does not wish to function or does not represent the majority of physicians in an area, the Secretary of HEW is required by law to designate another group as the PSRO agent.

We know nothing of the regions or regulations at this time. The regions are supposed to be determined by January 1, 1974. The regulations are supposed to be published by the spring of 1974.

If there is conformity after the guidelines are established, HEW will pay for services. If there is nonconformity, HEW will not authorize payment to providers, including hospitals and physicians.

If we desire to work within the law, there is much help available through committees of AMA, ASIM, AFMC, BC, BS, CPHS, and many computer companies.

The courses of action that are available now are:

1. Encourage repeal of the law—(PSRO section)—there is a bill in Congress now to try to accomplish this.

2. Actively oppose PSRO—however, HEW holds the purse strings and if physicians refuse to be involved, neither they nor the hospitals will be paid.

3. Encourage amending the law—this is the approach the AMA, ASIM, and AFMC are trying to follow, working through committees with HEW in pointing out the problem areas and working for amendment or different interpretations.

4. Take no action until the details are known and we as a group can decide whether, or how, we can function within the restrictions.

Our responsibilities as physicians

To guide our patients' care by determining all phases of medical necessity including admission, diagnosis, treatment, and level of care.

To provide the medical care and to guide the health care.

To do peer review and utilization—for only we can provide true peer review and assure any purchases of health care that his money is buying the best quality care.

The Foundation

KFMC was formed to provide a mechanism for Kansas physicians to provide peer review for Kansas, and to contract with third-party payors for medical care to do the peer review and to assure the maintenance of quality—not to become an HMO.

The Kansas Medical Society requested KFMC to do peer review for Kansas physicians.

The framework has been established to provide a working mechanism for state-wide coordinated, locally performed peer review for all third-party payors. However, we are unable to function as requested by KMS because it requires:

1. A great deal of money—The present source of money is through BC-BS, hospitals, the federal government, or donations by physicians. Currently, BC-BS and hospitals have money available to them from the federal government to provide these services (under their direction and control) including staff, forms, payment for physicians' peer review and utilization services, secretarial help, computer time, legal aid, publicity, etc. However, they can only contract with the designated PSRO agent to receive these federal funds.

2. And it requires a great deal of time. A great commitment of time will be necessary by all members of KMS and KOA. Time to perform the peer review for seven regional and 19 district committees will be necessary. A great deal of time will be required by all officers of KMS and KFMC, and a commitment by all Kansas physicians to guide and support the activities of KFMC.

At the request of KMS, KFMC supported the concept of one statewide PSRO region for Kansas. I indicated in testimony before HEW a great deal of support from many sections of health care services for this concept, as well as the ability of the Foundation to serve as the state PSRO agent. When the time comes to apply to become a PSRO agent, this action will have to be approved by a meeting of the corporate members of the Foundation—which in essence is the House of Delegates of KMS.

The results that may follow Congressional action on PSRO

If organized medicine accepts the possibility of guiding its own destiny, then we need to take the lead and provide the services that we alone can provide: determination of medical necessity, providing medical care, peer review, and guiding health services.

If we in organized medicine prefer not to determine our own future, then the control of medicine will go to some other body designated by HEW.

My recommendations are:

1. That at this time, no action be taken relative to any endorsement for or rejection of PSRO.

2. That we continue to work with all our power through organized medicine to try to modify the unworkable sections of the present PSRO law.

3. That the Foundation mechanism continue to be held in readiness to do the peer review and utilization. Also, that the Foundation not contract with any third party until specific details can be presented to you for your consideration and action.

4. That when we know the PSRO requirements, including the PSRO regions and guidelines and responsibilities, then KFMC will return to you with specific recommendations for your approval or rejection.

The final decision will be yours.

SECOND SESSION

After lunch, the House of Delegates reconvened for its second session.

Dr. Bill Roy explained the proposed Long-Ribicoff bill in the Congress. He stressed opposition to this bill.

The Reference Committee, composed of Dr. H. Thomas Gray, Wichita, Chairman; Drs. Evan Williams, Dodge City; Richard R. Beach, Topeka; Richard Gruendel, Kansas City, and Herman W. Hiesterman, Quinter, recommended adoption of the following resolutions.

RESOLUTION NO. F73-1

Medicredit Bill

WHEREAS, There are Americans who either because of their economic status or due to the nature of their

illness find the cost of health care to bring financial distress; and

WHEREAS, It appears that the federal government will institute some form of national health insurance; therefore be it

Resolved, That the Kansas Medical Society strongly urge that national health insurance provide a greater benefit for persons of lesser economic stature; and be it further

Resolved, That the AMA sponsored Medicredit Bill does this more equitably than any other legislation currently before Congress; and be it further

Resolved, That the Kansas Medical Society endorse and urge the passage of the Medicredit Bill as presently written; and be it further

Resolved, That copies of this resolution be sent to the Kansas Senators and Representatives.

RESOLUTION NO. F73-2

One PSRO For Kansas

Not adopted.

RESOLUTION NO. F73-3

Kansas Neurosurgical Society

WHEREAS, The neurosurgeons of Kansas wish to be recognized as a component organized specialty society of the Kansas Medical Society; therefore be it

Resolved, That the By-Laws be amended to add Section 4.58, 17, the "Kansas Neurosurgical Society."

RESOLUTION NO. F73-4

PSRO and HMO

Not adopted.

RESOLUTION NO. F73-5

Silver Nitrate

WHEREAS, Since the morbidity from gonorrheal ophthalmia neonatorum is substantial; and

WHEREAS, The incidence of gonorrhea in this state is on the increase; and

WHEREAS, The Credé's method of installation of silver nitrate drops in the newborn's eyes has been effective in preventing this disease; and

WHEREAS, The Section on Ophthalmology of the Kansas Medical Society voted unanimously to continue this method; therefore be it

Resolved, That no action be taken to change the legal status of this silver nitrate treatment, and that the Kansas Medical Society vigorously oppose any legislative attempt to change this status.

RESOLUTION NO. F73-6

Roster

WHEREAS, Resolution 73-20 was adopted by the House of Delegates on Wednesday, May 9, 1973, as follows:

Resolved, That the Roster be amended to include board certification, specialty training and specialty organization other than board certification attesting to clinical competence in accordance with guidelines adopted by the Kansas Medical Society, and be it further

Resolved, That the information on each physician be compiled by the component society in accordance with these guidelines; and

WHEREAS, Technical difficulties discovered in the executive office appear that it is impossible to produce a roster with meaningful information as suggested by the resolution; and

WHEREAS, One sizable component society said they would not supply us with this information; and

WHEREAS, Another sizable component society sent us four typewritten pages on one physician; and

WHEREAS, This would be a sizable cost because the type is retained at the printer and only those lines needing correction are altered. This would require an entire new typesetting; therefore be it

Resolved, That the House of Delegates declare Resolution 73-20 be rescinded, and that a committee be appointed to explore a practical way to provide the additional information that was intended by Resolution 73-20, and that the roster now be sent to the printer to appear as it did last year.

RESOLUTION NO. F73-7

PSRO

Not adopted.

RESOLUTION NO. F73-8

Non-Physician Individuals Rendering Medical Care

Combined with No. F73-10.

RESOLUTION NO. F73-9

Postgraduate Education

Not adopted.

RESOLUTION NO. F73-10

Paramedical Personnel

WHEREAS, The increased use of paramedical personnel by physicians requires the Society to take a stand on this subject; therefore be it

Resolved, That it is the position of the Kansas Medical Society that paramedical personnel not be licensed because they are working under the direction and supervision of licensed physicians; and be it further

Resolved, That billing for services rendered by paramedical personnel is the responsibility of the licensed physician and not the responsibility of persons who do not have a license to perform independent professional services; and be it further

Resolved, That issues of conflict regarding the use of paramedical personnel be resolved by the area peer review committees under the Kansas Foundation for Medical Care.

RESOLUTION NO. F73-11

Probationary Members

Not adopted.

RESOLUTION NO. F73-12

Smallpox Vaccination

WHEREAS, It is recommended by the American Academy of Pediatrics and the National Institute of Health that mass smallpox vaccination be discontinued; and

WHEREAS, The State of Kansas Statutes continue to require all children entering school to be vaccinated; and

WHEREAS, Physicians are therefore required by law to act contrary to those recommendations or justify an alternative action; therefore be it

Resolved, That the Kansas Medical Society exert its influence to:

1. Rescind Kansas statutes requiring smallpox vaccination;
2. Allow physicians to use their professional judgement regarding special circumstances in which smallpox vaccination might be indicated.

RESOLUTION NO. F73-13

Combined with No. F73-10.

RESOLUTION NO. F73-14

A Code of Trust Between Hospitals and Their Patients

Not adopted.

RESOLUTION NO. F73-15

PSRO

Not adopted.

RESOLUTION NO. F73-16

Family Health Insurance

WHEREAS, The concept of family health insurance to protect a family from unusual medical expenses which are actuarily predictable is sound; and

WHEREAS, In this state it is possible to purchase a "family" policy which excludes newborns and children until a required waiting period has passed; and

WHEREAS, The treatment of newborn disease and congenital disease has become increasingly sophisticated, successful and expensive; therefore be it

Resolved, That the Kansas Medical Society:

1. Supports the concept of family health insurance as a method of payment of unusual medical expenses for the entire family;
2. Supports an effort to enact legislation to prevent the sale of health insurance policies which exclude newborns.

RESOLUTION NO. F73-17

Kansas Relative Value Schedule

Resolved, That the Kansas Medical Society reaffirms Resolution 70-38; and be it further

Resolved, That the 3rd Edition AMA-CPT shall be the official nomenclature for the current Kansas Relative Value Schedule.

RESOLUTION NO. F73-18

Healing Arts Act

WHEREAS, Visiting professors come to hospitals within the state for educational purposes; and

WHEREAS, These national and international authorities are especially skilled in certain procedures; and

WHEREAS, Not to perform and demonstrate to students of medicine (undergraduate, graduate, and

postgraduate) these procedures, in part, prevents fulfillment of their purpose of coming to that institution; and

WHEREAS, Under the current Board of Healing Arts Act, they are not legally permitted to accept primary responsibility for a procedure; and

WHEREAS, This is not in the best interest of education within our state; therefore be it

Resolved, That an amendment to the Board of Healing Arts Act to allow temporary licensure for visiting professors properly authenticated and in our state for purposes of education is supported by the Kansas Medical Society. This temporary licensure should be accomplished in an appropriate manner but sufficiently simplified to not impair the utilization and acquisition of said visiting professors with such expertise.

RESOLUTION NO. F73-19

Reorganization of Kansas State Board of Health

Resolved, That the existing structure of the Kansas State Board of Health and the Kansas State Department of Health need change to improve efficiency and better meet the expanding problems and needs of the state regarding health and the environment; and be it further

Resolved, That the present organizational structure, with appropriate modifications, can meet these needs more adequately and economically than complete departmental reorganization as called for in various legislative committee reports.

RESOLUTION NO. F73-20

Secretary of State Department of Social Welfare and Rehabilitative Services

Not adopted.

RESOLUTION NO. F73-21

Blue Shield Participating Physicians

Not adopted.

RESOLUTION NO. F73-22

Allied Health Professionals

Resolved, That it is the position of the Kansas Medical Society that no additional license privileges be granted allied health professionals to act as independent practitioners.

RESOLUTION NO. F73-23

Lt. General Richard R. Taylor, M.D.

WHEREAS, The State of Kansas has been honored by the selection of Lt. General Richard R. Taylor, M.D. as Surgeon General of the United States Army; therefore be it

Resolved, That Lt. General Richard R. Taylor, M.D. be invited to speak to the Kansas Medical Society House of Delegates at the Spring Meeting of 1974.

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INDEX TO VOLUME LXXIV

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INDEX TO VOLUME LXXIV

Calendar Year 1973

Authors

Anderson, Larry 306
Asher, Marc A. 91
Beard, James D. 1
Behbehani, Abbas M. 109
Bichlmeier, Franklin G. 297, 411
Bolton, Merle R., Jr. 394
Bouda, David 87
Bruner, Steven C. 201
Burch, Timothy K. 335
Burnett, A. Dean 329
Byrne, Robert F. 265
Carter, Thomas N. 138
Clark, Anna E. 96
Clark, Courtney 446
Clough, Charles A. 190
Collins, Harold A. 273
Covell, James W. 303
Croshy, Ivan K. 226
Crouch, John A. 78
Dick, Arthur 87
Diederich, Dennis A. 406
Diehl, Antoni M. 78, 106
Dobbs, William H. 12
Drevets, Curtis C. 33
Egbert, Barry 72
Ellis, Lee 310
Ensandi, Ali A. 46
Fast, Robert E. 459
Felt, Samuel E. 43
Fink, Robert D. 1
Fitzgerald, Thomas J. 456
Fortin, Don 367
Franzen, Irvin G. 310
Frazier, R. L. 129
Galvan, Alonso 361
Givner, David 265
Gonzalez, Giomar 224, 361
Grantham, Jared J. 406
Graves, J. W. 332
Gray, Wayne 394
Greenwood, Ronald D. 135, 220, 301, 339
Grossmann, A. Eugene 423
Grotheer, Martin H. 142
Guillan, Ramon A. 217
Hardin, Creighton A. 406
Head, Murdock 9
Hermreck, Arlo S. 197, 371, 406
Hirsch, Martin S. 280
Hoofer, W. D. 329
Jacobs, David S. 43
Jacobs, Rae R. 85, 101
Jagger, Paul I. 304
Jewell, W. R. 129
Kelly, Charles R. 190
Kentsmith, David K. 138
Kepes, John 441
Kepes, Magda 82
Killen, Duncan A. 273
Klotz, Arthur P. 423
Knapp, M. Robert 446
Knott, David H. 1
Korb, Thomas 423
Lawson, Dwight 343, 463
Lerner, Scott A. 194, 276
Lewis, H. Daniel 394
Luckstead, Eugene F. 335
Mann, Nirmal S. 269
Maxwell, John A. 190
McReynolds, Charles R. 399
Mebust, Winston K. 406
Mershon, James C. 450
Miller, Don R. 369
Morris, John A. 96
Murphy, Daniel 82
Nagaswami, Subramoney 441
Neuenschwander, John Rand 228
Owen, LaRue W. 446
Owen, Pere A. 446
Pereira, Willy 450
Pierleoni, Robert G. 68
Pilchard, William A. 115
Porter, Robert D. 406
Pro, John D. 367
Rappolt, Richard T., Sr. 15
Reckling, Fred W. 190, 197, 371
Reed, William A. 226
Reimer, Donald R. 456
Rengachary, Setti S. 82
Rhodes, James B. 423
Rhodes, Robert J. 103
Ryan, Michael 72
Schuman, Norvin D. 185
Schwegler, Raymond 394
Sethi, Gulshan K. 224
Shah, Sharfuddin 399
Sosnow, William 85
Stanley, W. A. 106
Stavros, George E. 16
Steinzeig, Sherman M. 297, 411
Stoffer, Robert P. 399
Stratton, Richard L. 48
Taub, Arnold E. 135
Tayiem, A. K. 332
Thomas, Thomas V. 297, 411
Tillema, David A. 91
Toalson, William B. 297, 411
Traisman, Howard S. 135
Tucker, Donald R. 406
Twemlow, Stuart W. 441
Van Dyke, Peter C. 135
Vatner, Stephen F. 140
Vera-Roman, Jose M. 369
Villanueva, Cesar 38
Wallace, Robert 446
Wathanacharoen, Suchint 226
Yang, Chih-Ping 418
Zelman, Samuel 217
Ziegler, Dewey K. 72

Index to Subject Matter

Allergy and immunology:
- cardiac transplantation: immunosuppressive role of anticoagulation 226
- myasthenia gravis and immunity 72
- urticaria and angio-edema 33

American College of Physicians, Kansas Chapter, issue:
- atrial arrhythmia, unusual: multifocal atrial tachycardia .. 394
- coronary surgery, practical: aorto-coronary saphenous vein bypass graft 411
- glucose tolerance tests: routine assay of insulin 399
- persistent ventricular tachycardia: the use of Diphenylhydantoin 418
- reflux esophagitis 423
- renal transplantation 406

American College of Surgeons, Kansas Chapter, issue:
- splenic cysts 332
- thoracic outlet relief: first rib excision 329

American Medical Association: actions of house of delegates 57, 349

Appendages (see Skin)

Blood and lymphatics system:
- cardiac transplantation: immunosuppressive role of anticoagulation 226
- Hodgkin's, infections and neutrophils 185
- mystery virus: the elusive E-B virus 109
- rheumatic fever evaluation 106
- splenic cysts 332

Cardiovascular system:
- aortic valve replacement: prosthesis related complications 273
- atrial arrhythmia, unusual: multifocal atrial tachycardia 394
- cardiac:
  - complications: periarteritis nodosa of infancy 78
  - transplantation: immunosuppressive role of anticoagulation 226
- cardiomyopathy, functional obstructive: familial idiopathic hypertrophic subaortic stenosis in children 335
- coronary surgery, practical: aorto-coronary saphenous vein bypass graft 411
- midsystolic clicks and late systolic murmurs 276
- myocardial failure: ventricular aneurysm and adynamic areas 297
- pericardial cyst 369
- persistent ventricular tachycardia: the use of Diphenylhydantoin 418
- Raynaud's disease 228
- rheumatic fever evaluation: secondary prophylaxis program 106
- splenic arteriovenous fistula 224
- thoracic outlet relief: first rib excision 329

Dermatology:
- actinic keratosis: the advent of Fluorouracil 306
- incontinentia pigmenti 43
- neurofibromatosis with sarcoma 194
- Waardenburg's syndrome 301

Emergency care:
- new developments 85
- stab wounds of the head, neck, and thorax, multiple 371

Endocrine system:
- glucose tolerance tests: routine assay of insulin 399

Endocrinology:
- glucose tolerance tests: routine assay of insulin 399
- growth patterns and diabetes 135

Family practice:
alcoholism ... 1
streptococcal pharyngitis ... 201
Gastrointestinal system:
gastroduodenal visualization: fiberoptic esophagogastroduodenoscopy ... 269
glucose tolerance tests; routine assay of insulin ... 399
growth patterns and diabetes ... 135
non-operative visualization: per-oral transduodenal pancreatography and cholangiography ... 361
reflux esophagitis ... 423
Genetics:
neurofibromatosis with sarcoma ... 194
Waardenburg's syndrome ... 301
Internal medicine:
alcoholism ... 1
aortic valve replacement ... 273
atrial arrhythmia, unusual: multifocal atrial tachycardia .. 394
cancer prognosis ... 129
cardiac transplantation: immunosuppressive role of anticoagulation ... 226
cardiomyopathy, functional obstructive: familial idiopathic hypertrophic subaortic stenosis in children ... 335
CODAC: community organization for drug abuse control .. 16
coronary surgery, practical: aorto-coronary saphenous vein bypass graft ... 411
drug abuse ... 9
gastroduodenal visualization: fiberoptic esophagogastroduodenoscopy ... 269
glucose tolerance tests: routine assay of insulin ... 399
growth patterns and diabetes ... 135
Hodgkin's, infections and neutrophils ... 185
methadone programs ... 12
midsystolic clicks and late systolic murmurs ... 276
multiple sclerosis ... 87
myocardial failure: ventricular aneurysm and adynamic areas ... 297
mystery virus: the elusive E-B virus ... 109
narcotic antagonist: sublingual injection in acute heroin overdose ... 15
non-operative visualization: per-oral transduodenal pancreatography and cholangiography ... 361
pericardial cyst ... 369
persistent ventricular tachycardia: the use of Diphenylhydantoin ... 418
Pseudomonas pneumonia ... 280
pulmonary Nocardia asteroides: successful treatment with Sulfadiazine ... 265
Raynaud's disease ... 228
reflux esophagitis ... 423
rheumatic fever evaluation: secondary prophylaxis program 106
streptococcal pharyngitis ... 201
Sydenham's chorea ... 459
thoracic pseudomyxoma ... 217
Kansas Medical Society:
annual session:
alcoholism ... 1
CODAC: community organization for drug abuse control 16
drug abuse ... 9
methadone programs ... 12
narcotic antagonist: sublingual injection in acute heroin overdose ... 15
council minutes ... 211, 258, 388
house of delegates, report of ... 239
KUMS:
issue (see University of Kansas School of Medicine)
trauma conference:
shotgun wound of the abdomen, point-blank ... 197
stab wounds of the head, neck and thorax, multiple ... 371
Laboratory medicine (see Pathology)
Lymphatics (see Blood)
Medical history:
Abt, Isaac Arthur ... 376
Brenneman, Joseph ... 339
Musculo-skeletal system:
carpal tunnel syndrome ... 190
low back pain ... 101
myasthenia gravis and immunity ... 72
Perthes' disease ... 91
spondylitic cord compression: with focal destructive vertebral lesions ... 82
thoracic outlet relief: first rib excision ... 329
Nervous system:
cerebellar dyssynergia, progressive: the use of diazepam 138
homosexual aversion therapy: electric shock technique ... 103
marijuana by medical students, the use of ... 142
myasthenia gravis and immunity ... 72
mycoplasma infection, human ... 441
neurofibromatosis with sarcoma ... 194
operant conditioning procedures: conditioning in schizophrenic work ... 456
psychiatry, physicians and ... 367
spondylitic cord compression: with focal destructive vertebral lesions ... 82
Sydenham's chorea ... 4
Neurology (see Psychiatry)
Obstetrics and gynecology:
fetal monitoring ... 96
monitoring, the value of: fetal asphyxia and fetal surveillance ... 38
Oncology:
cancer prognosis ... 129
Hodgkin's, infections and neutrophils ... 185
multicentricity in cancer of the breast ... 48
neurofibromatosis with sarcoma ... 194
Ophthalmology:
quantitative perimetry: visual field assessment ... 115
Waardenburg's syndrome ... 301
Orthopedics:
carpal tunnel syndrome ... 190
low back pain ... 101
Perthes' disease ... 91
spondylitic cord compression: with focal destructive lesions 82
Otolaryngology:
streptococcal pharyngitis, diagnosis of ... 201
Waardenburg's syndrome ... 301
Pathology and laboratory medicine:
glucose tolerance tests: routine assay of insulin ... 399
Hodgkin's infections and neutrophils ... 185
multiple sclerosis ... 87
mycoplasma infection, human ... 441
mystery virus: the elusive E-B virus ... 109
Pediatrics:
Abt, Isaac Arthur ... 376
Brenneman, Joseph ... 339
cardiac complications: periarteritis nodosa of infancy ... 78
cardiomyopathy, functional obstructive: familial idiopathic hypertrophic subaortic stenosis in children ... 335
growth patterns and diabetes ... 135
psychiatry in pediatrics ... 220
rheumatic fever evaluation: secondary prophylaxis program 106
Pharmacology and therapeutics:
actinic keratosis: the advent of Fluorouracil ... 306
alcoholism ... 1
cerebellar dyssynergia, progressive: the use of diazepam 138
CODAC: community organization for drug abuse control 16
drug abuse ... 9
marijuana by medical students, the use of ... 142
methadone programs ... 12
narcotic antagonist: sublingual injection in acute heroin overdose ... 15
persistent ventricular tachycardia: the use of Diphenylhydantoin ... 418
pulmonary Nocardia asteroides: successful treatment with Sulfadiazine ... 265
Physiology:
fetal monitoring ... 96
monitoring, the value of: fetal asphyxia and fetal surveillance ... 38
Preventive medicine:
rheumatic fever evaluation: secondary prophylaxis program 106
Psychiatry and neurology:
alcoholism ... 1
cerebellar dyssynergia, progressive: the use of diazepam .. 138
CODAC: community organization for drug abuse control .. 16
drug abuse ... 9
homosexual aversion therapy: electric shock technique ... 103
methadone programs ... 12
multiple sclerosis ... 87
myasthenia gravis and immunity ... 72
narcotic antagonist: sublingual injection in acute heroin overdose ... 15
operant conditioning procedures: conditioning in schizophrenic work ... 456
pediatrics, psychiatry in ... 220
physicians and psychiatry ... 367
quantitative perimetry: visual field assessment ... 115
spondylitic cord compression: with focal destructive vertebral lesions ... 82
Public health:
CODAC: community organization for drug abuse control 16
drug abuse ... 9
methadone programs ... 12
Radiology and nuclear medicine:
non-operative visualization: per-oral transduodenal pancreatography and cholangiography ... 361
Reproductive system: fetal monitoring ... 96
Respiratory system:
cancer prognosis ... 129
monitoring, the value of: fetal asphyxia and fetal surveillance ... 38
Pseudomonas pneumonia ... 280
pulmonary Nocardia asteroides: successful treatment with Sulfadiazine ... 265
streptococcal pharyngitis ... 201
thoracic pseudomyxoma ... 217
Skin and appendages system:
actinic keratosis: the advent of Fluorouracil ... 306
incontinentia pigmenti ... 43
multicentricity in cancer of the breast ... 48
neurofibromatosis with sarcoma ... 194
urticaria and angio-edema ... 33
Waardenburg's syndrome ... 301
Socio-economic medicine and health care delivery:
educational approach: teaching health care professionals .. 68
disability insurance under Social Security ... 343

minor surgery center 446
Supplemental Security Income Program 463
Williams-Steiger Act: occupational safety and health in Kansas 310
Special senses:
quantitative perimetry: visual field assessment 115
Waardenburg's syndrome 301
Student theses:
actinic keratosis: the advent of Fluorouracil 306
marijuana by medical students, the use of 142
multicentricity in cancer of the breast 48
Pseudomonas pneumonia 280
Raynaud's disease 228
streptococcal pharyngitis, diagnosis of 201
Sydenham's chorea 459
Surgery:
aortic valve replacement: prosthesis related complications .. 273
cardiac transplantation: immunosuppressive role of anti-coagulation 226
coronary surgery, practical: aorto-coronary saphenous vein bypass graft 411
carpal tunnel syndrome 190
minor surgery center 446
pericardial cyst 369
renal transplantation 406
splenic:
arteriovenous fistula 224
cysts 332
stab wounds of the head, neck, and thorax, multiple 371
thoracic:
outlet relief: first rib excision 329
pseudomyxoma 217
Therapeutics (see Pharmacology)
University of Kansas School of Medicine, issue:
cardiac complications: periarteritis nodosa of infancy 78
educational approach: teaching health care professionals .. 68
emergency care 85
fetal monitoring 96
homosexual aversion therapy: electric shock technique ... 103
low back pain 101
multiple sclerosis 87
myasthenia gravis and immunity 72
mystery virus: the elusive E-B virus 109
Perthes' disease 91
quantitative perimetry: visual field assessment 115
rheumatic fever evaluation: secondary prophylaxis program 106
spondylitic cord compression: with focal destructive vertebral lesions 82
Urinary system: renal transplantation 406
Urology: renal transplantation 406

Editorials

Beneficent subsidy, The 208
Benefit performance 384
Dilemma *du jour* 466
Fable 120
Medical article, The 19
Perplexing power, This 146
Record, For the 426
Rural practice: what health professionals look for 234
Sibling rivalry 317
Spinal analysis, In the 54
Truth shall make your fee, The 346
Whose truth goes marching on 286

Obituaries

Collins, Harold L. 289
Cox, Michael J. 348
Fuller, Clinton C. 289
Glassen, Mary 56
Grieve, George H. 289
Hamilton, James T. 178
Haughey, Leo E. 56
Hertzler, John W. 56
Hoover, Charles O. 319
Isaacs, Fred R. 178
King, Harry W. 390
Krahenbuhl, Marcella M. 468
Krehbiel, Bertrand I. 348
Love, William A. 178
Maggard, Delano I. 178
McNickle, Jerry H. 178
Moore, Robert H. 56
Munger, C. Herbert 289
Orrison, William W. 390
Patton, George A. 319
Proctor, Lawrence A. 348
Schwartz, Willard C. 468
Shonyo, Elwin S. 390
Steffen, Marvin O. 390
Titus, Albert E. 468
Wheeler, J. Albert 319
Williams, Harold O. 468

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